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THE GALACTOSE TOLERANCE TEST IN THYREOTOXICOSIS.[1]

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Carbohydrate Metabolism in Thyreotoxicosis.

THE deranged carbohydrate metabolism of thyreotoxicosis has for many years interested both the physicians and the biochemists; but latterly until the introduction of the galactose test this interest has tended to wane.

This subject was first approached by Cramer and Krause,[(1)] in 1913, who demonstrated that thyreotoxicosis induced in rats and cats resulted in great depletion of the hepatic glycogen despite a diet rich in carbohydrate. This has subsequently been confirmed for laboratory animals by many authors, including Coggeshall and Greene,[(2)] and hepatic glycogen depletion has been repeatedly described in cases of thyreotoxicosis.

By respiratory quotient determinations Sanger and Hun[(75)] were able to show that although a high blood sugar level was reached in thyreotoxic patients after the ingestion of glucose, there was increased utilization of carbohydrate by these patients when compared with normal subjects; they concluded that the high blood sugar levels indicated an inability to store glucose, most probably a failure of liver storage due to some toxic change in the liver. Johnston,[(44)] too, has concluded that oxidation of carbohydrate in patients suffering from hyperthyreoidism occurs more readily than in normal persons.

Woodyatt, Sansum and Wilder[(90)] introduced glucose intravenously at a constant rate and estimated the degree of the resulting glycosuria. From this they concluded that there was a lowered carbohydrate tolerance in exophthalmic goitre; but, as will be mentioned below, it is possible that the fate of carbohydrate introduced into a peripheral vein differs from that of carbohydrate entering the body by the portal circulation. A deduction of impaired tolerance based solely on glycosuria, as in these experiments, is almost certainly erroneous, for the excretion of some of the carbohydrate by the kidneys would probably occur before the carbohydrate reached the liver.

Althausen and Stockholm[(3)] have shown that there is an increased rate of absorption of glucose from the bowel in thyreotoxic animals, and they suggested that the thyreoid hormone influences absorption mainly by accelerating phosphorylation in the intestinal mucosa and by stimulating gastric emptying. The intestinal absorption of the sugars, glucose and galactose, at a rate greater than that accounted for by simple diffusion, is due to the conjugation of the sugar and phosphate radicle by phosphatase in the mucosal cells to form hexose phosphate which, after rapid passage through the cells, liberates the hexose into the peripheral circulation. It has been suggested (Thorpe[(83)]) that this process of phosphorylation is also under the control of the adrenal cortical hormone.

With these suggestions of Althausen and Stockholm in mind, opaque meals have been given to the last eight thyreotoxic patients admitted to Prince Henry Hospital, and skiagrams have been taken. All showed rapid emptying of the stomach, and in most cases the barium had reached the colon within one and a half hours. Crotti[(22)] found that the stomachs of thyreotoxic patients were often in a state of constant spasticity, as seen fluoroscopically; but in our few patients the condition was rather one of accelerated peristalsis than of spasm. Hypochlorhydria and achlorhydria have been frequently reported in thyreotoxicosis by many authors, including Friedenwald, Morrison and Morrison,[(29)] Lerman and Means,[(50)] Moll and Flint,[(66)] Moll and Scott,[(64)] Moore,[(67)] and Wilkinson.[(97)] In eight cases of thyreotoxicosis, fractional test meal examinations (Ewald) have revealed achlorhydria; but in each

[1] Read at a library seminar at Prince Henry Hospital, Sydney, on July 14, 1941.

case it was impossible to obtain a specimen for examination forty or sixty minutes after the meal, owing apparently to the rapidity of gastric emptying. Within a fortnight of operation the hypermotility of the bowel, as shown by the opaque meal examination, decreases. The increased peristalsis of thyreotoxicosis, by rapidly moving the food to the absorptive region of the bowel, probably accelerates intestinal absorption.

Crawford (1940)[21] has suggested that the increased secretion of adrenaline in thyreotoxicosis probably plays a part in rapidly mobilizing the liver glycogen immediately it is deposited, thus preventing its accumulation.

From a consideration of these experiments it appears that impaired tolerance is not a sufficiently comprehensive term to apply to the accelerated intestinal absorption, the rapid mobilization of hepatic glycogen and the increased utilization of glucose by the tissues in thyreotoxicosis; but no other term has yet been suggested to cover the changes in carbohydrate metabolism of these patients. Again, in hypothyreoidism the "flat" glucose tolerance curve has been referred to in the past as indicative of increased tolerance; but in a condition in which the other functions of the body are retarded it is not to be expected that carbohydrate metabolism alone would be accelerated. Crawford (1940),[21] by intravenous glucose-tolerance tests, has shown that in hypothyreoidism the low blood sugar curves are due to slow absorption of the carbohydrate from the bowel. Similarly, Crawford (1939),[20] Ross,[72][73] Ross and Tonks[74] and Lepore[64] have shown that the "flat" oval glucose tolerance curves of patients suffering from abdominal tuberculosis and cœliac disease are due to impaired absorption. While this is the expected result in these cases associated with extensive intestinal lesions, and while these "flat" curves are no longer referred to as showing "improved tolerance", yet in myxœdema, in which intestinal derangement though less obvious is nevertheless present, the term "improved or increased tolerance" still persists.

Although the utilization of glucose by the tissues is accelerated in thyreotoxicosis, Griffiths[32] and other authors have shown that a condition of insulin resistance exists in this disease.

Kugelmann[33] found elevated blood sugar curves after the administration of lævulose to thyreotoxic patients, and concluded that the underlying cause was an inability on the part of the liver to change the lævulose to glucose and to store it.

John (1926)[40] found the fasting blood sugar level raised in only six out of 68 cases of thyreotoxicosis, whereas the blood sugar curve was elevated in all his cases. Most authors report similar changes in the blood sugar curve in thyreotoxicosis; but it would appear that a slight increase of the fasting blood sugar to the region of 110 milligrammes *per centum* is a common finding, and at Prince Henry Hospital we have found such an elevation in most of our cases. The average fasting blood sugar level of our thyreotoxic patients this year has been 108 milligrammes per 100 millilitres of blood; but none of these patients has shown glycosuria while fasting (the patient with frank diabetes is not included in this group).

Youman and Warfield[31] reported a series of thyreotoxic patients, 22 out of 27 of whom showed a "decreased sugar tolerance". The average fasting blood sugar content of their patients was 107·4 milligrammes *per centum*.

The question of the diagnosis of diabetes complicating thyreotoxicosis is a difficult one. Joslin and co-workers[46][47] have suggested a fasting blood sugar level of 150 milligrammes *per centum* and a postprandial blood sugar level of 200 milligrammes *per centum* as the standard for this diagnosis, and in only one patient this year have we found the fasting blood sugar to be above this level. This patient gave a typical diabetic curve after the ingestion of 50 grammes of glucose (Figure I), but was only mildly thyreotoxic. Another patient, aged seventy years, with a toxic adenomatous thyreoid gland and a fasting blood sugar level of only 102 milligrammes *per centum* showed a blood sugar curve falling slowly from its peak (Figure II), and possibly she should be classed as mildly diabetic. It is of interest that two of her brothers had died from diabetes, but neither had suffered from thyreotoxicosis. Joll, in his book "Diseases of the Thyroid Gland",[48] gives such a curve as this as typical of diabetes complicating thyreotoxicosis.

TABLE I.
Glucose Tolerance Tests on Thyreotoxic Patients.

Case Number.	Fasting Blood Sugar Level. (Milligrammes *per centum*.)	Renal Threshold. (Milligrammes *per centum*.)	Blood Sugar Peak. (Milligrammes *per centum*.)	Glycosuria. (Percentage.)	Basal Metabolic Rate. (Percentage above Normal.)
I	96	180	194	0·5	27
II	165	172	180	Trace	26
III	102	176	232	0·2	47
IV	93	(165)[1]	165	Nil	43
V	93	—	136	—	22
VI	93	(194)[1]	194	Nil	76
VII	112	168	180	1·3	54
VIII	96	(172)[1]	172	Nil	66
IX	100	(224)[1]	224	Nil	19
X	122	(223)[1]	223	Nil	54
XI	136	184	232	4·0	56
XII	135	197	210	0·6	35
XIII	102	202	256	0·5	50
XIV	72	(226)[1]	226	Nil	66
XV	82	168	254	1·5	14
XVI	115	—	230	2·0	35
XVII	125	218	296	0·5	14
XVIII	84	(228)[1]	228	Nil	47
XIX	114	—	200	2·0	37
XX	136	(234)[1]	234	Nil	52
XXI	84	194	216	Trace	62
XXII	100	221	233	0·7	41
XXIII	106	172	216	1·0	24

[1] During these tests the blood sugar levels did not reach the renal thresholds.

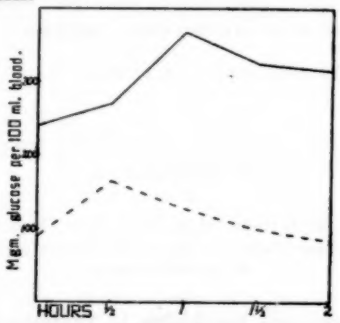

FIGURE I.
Solid line: blood glucose curve from Case XXXIX, *diabetes mellitus* with mild thyreotoxicosis. Interrupted line: average normal glucose curve.

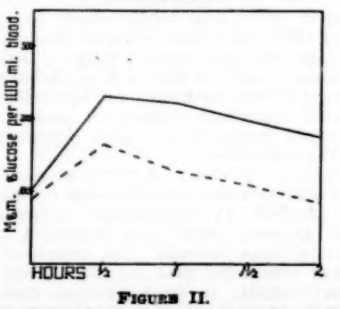

FIGURE II.
Solid line: blood glucose curve from Case III, severe thyreotoxicosis supervening on an adenomatous thyreoid with possible mild *diabetes mellitus*. Interrupted line: average normal glucose curve.

Joslin and Lahey[46] found that after the ingestion of 50 grammes of glucose 38·6% of patients with primary thyreotoxicosis showed glycosuria. In cases of secondary thyreotoxicosis the incidence of glycosuria was 27·7%. On their criterion of a fasting blood sugar level of 150

milligrammes *per centum* these authors classified only 2·5% of their thyreotoxic patients as diabetic.

From an investigation of several thousand cases John (1932)[43] reported the average incidence of hyperthyreoidism among cases of diabetes as 1·68%, and the incidence of diabetes in hyperthyreoid patients as 2·31%; but Fitz[21] thinks that the association is probably due to chance.

A thyreotoxic patient now in Dr. Poate's clinic at the Prince Henry Hospital illustrates the difficulty in diagnosis encountered by practitioners in districts in which ancillary tests, such as the glucose tolerance test and the basal metabolic rate estimation, are not available. This patient's thyreoid gland had been slightly enlarged for the past forty years, but for the past two years he had been treated as a diabetic because of loss of weight and glycosuria. However, his glucose tolerance curve (Figure III) was of the type commonly found in thyreotoxicosis,

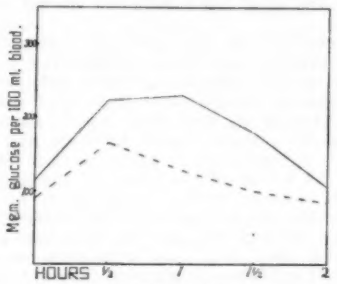

FIGURE III.

Solid line: blood glucose curve from Case XVI, severe thyreotoxicosis; the type of glucose curve commonly found in thyreotoxicosis. Interrupted line: average normal glucose curve.

definite thyreotoxic symptoms were elicited when he was questioned, and the basal metabolic rate was +35%. He also presented an elevated blood galactose curve, which, as will be mentioned below, has been shown to be confirmatory evidence, if such is necessary in this case, of the presence of thyreotoxicosis and not of diabetes.

Although the majority of glucose tolerance curves in thyreotoxicosis are of the type illustrated in Figure III, other curves are occasionally obtained, the shape of which does not conform to the typical curve. As already mentioned (Figures I and II), the glucose tolerance curve differs from the typical curve if thyreotoxicosis develops

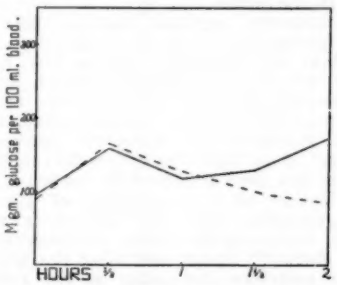

FIGURE IV.

Solid line: blood glucose curve from Case VIII, severe thyreotoxicosis; an irregular type of curve. Interrupted line: average normal glucose curve.

in a diabetic or if a latent diabetes is aggravated by the development of thyreotoxicosis. Another type of glucose curve less commonly found in thyreotoxicosis is shown in Figure IV. The rapidity with which restoration towards normality occurs after operation is astonishing and is shown in Figure V. This patient had a subtotal thyreoidectomy performed by Dr. Hugh R. G. Poate one week after the first test and one week before the second. A similar change towards normality after thyreoidectomy has been found with the other types of curve.

Figure VI shows the alteration in the glucose tolerance curve produced in a myxœdematous patient by two weeks'

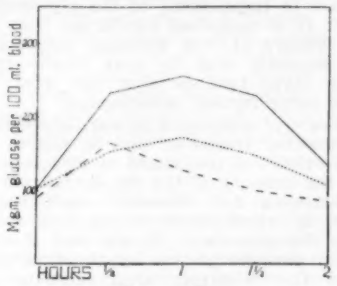

FIGURE V.

Solid line: blood glucose curve from Case XIII (thyreotoxicosis), one week prior to operation. Dotted line: glucose curve from same case one week after subtotal thyreoidectomy. Interrupted line: average normal glucose curve.

treatment with three milligrammes of thyroxin per day, given orally. During these two weeks her basal metabolic rate increased by from +26% to +18%. When she was first examined this patient's glucose tolerance curve was within

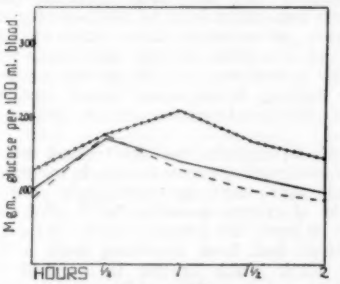

FIGURE VI.

Solid line: blood glucose curve from Case XXXVII, post-operative myxœdema treated with thyreoid extract. Interrupted line with rings: blood glucose curve from the same case after two weeks' treatment with three milligrammes of thyroxin per day. Interrupted line: average normal glucose curve.

normal limits; but this may be accounted for by the fact that she had been taking one grain of thyreoid extract three times a day for many months beforehand. The typical "flat" glucose tolerance curve of an untreated myxœdematous patient is illustrated in Figure VII.

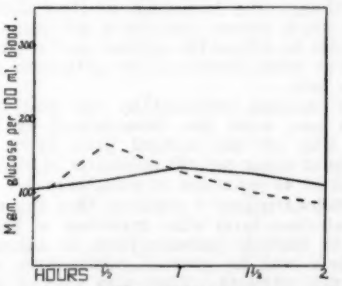

FIGURE VII.

Solid line: blood glucose curve from Case XXIV, myxœdema. Interrupted line: average normal glucose curve.

From a study of this small series of glucose tolerance curves the view of John (1927),[43] that no curve is diagnostic of thyreotoxicosis, is substantiated.

The variable delay in the fall of the curve from its peak, which is found in some cases and which, when it is considerable, as in Figure II, may lead to a diagnosis of diabetes complicating thyreotoxicosis, is probably related in part to impairment of the glycogenic function of the liver. It is doubtless significant that the patient from whom Figure II was obtained excreted only 0·81 gramme of hippuric acid in four hours—that is, the result of her liver function test was 27% of normal. This point is being further investigated.

The occurrence of glycosuria in our thyreotoxic patients has been somewhat irregular, and in degree it has not been directly related to the height of the glucose tolerance curve or to the severity of the thyreotoxicosis (Table I). Robinson, Derivaux and Hewell[69] have suggested the participation of phosphorylation in the resorption of glucose from the glomerular filtrate, and it may be that stimulation of phosphorylation by the thyreoid hormone accounts for the relatively small amount of glucose present in the urine of thyreotoxic patients compared with the height of the glucose tolerance curve and for their elevated renal threshold for glucose. The renal thresholds of 23 of our thyreotoxic patients are shown in Table I.

The case reported by Womack and Cole[69] is of interest. Instead of the high glucose curve so frequently obtained in thyreotoxicosis, their patient has a fasting blood sugar level of 72 milligrammes *per centum* and a blood sugar peak of only 117 milligrammes *per centum*, even when his basal metabolic rate was +42%. Because of this anomalous finding they suggested that in this case the thyreotoxicosis was secondary to overactivity of the islets of Langerhans; accordingly they removed the tail and greater part of the body of the pancreas, with cure of the thyreotoxic symptoms. On the patient's discharge from hospital his fasting blood sugar level was 80 and the peak of the glucose tolerance curve 235 milligrammes *per centum*.

It is difficult to explain the "flat" blood sugar curve in this case, for although thyreotoxicosis is a common disease, such a glucose curve must be exceedingly rare in this condition. It is of course possible by a diet very rich in carbohydrate to lower the glucose curve; but even assuming that the patient had been receiving such a diet, we are still in the dark when trying to explain the clinical improvement that followed partial pancreatectomy.

Metabolism of Galactose.

For many years American and German biochemists have studied the metabolism of galactose in normal and diabetic subjects and in laboratory animals; but the literature on this subject has been rather limited. Some of these papers will be briefly reviewed. From the results of their experiments it would appear that galactose absorption from the bowel depends on the process of phosphorylation, that galactose as such is not utilized by the tissues, that the metabolism of galactose is independent of insulin, that conversion of galactose to glucose occurs in the liver, and that although the formation of liver glycogen from galactose is much slower than from glucose, the body as a whole is able to utilize the greater part of the galactose ingested. The renal threshold for galactose, if it exists, is extremely low.

The blood changes produced by the oral ingestion of galactose in man were first investigated by Kahler and Machoid,[48] who set the normal limit as a rise of 30 milligrammes of sugar per 100 millilitres of blood following the ingestion of 40 grammes of galactose.

Roe and Schwartzman[71] reported that the elevation of the blood galactose level after ingestion of galactose was no greater in diabetic patients than in normal subjects, and that there was no greater galactosuria in diabetics than in normal subjects. They also showed that insulin did not affect the metabolism of galactose in the laboratory animal, and because of the apparently normal galactose tolerance of diabetics they suggested that galactose might be a valuable adjunct to the diet of diabetic patients. Deuel, Gulick and Butts[28] have reported that galactose has a higher antiketogenic value than glucose. In this connexion it is to be noted that the ruling price for galactose is 76s. (Australian) per pound, but that Roe and Schwartzman[71] had their galactose donated to them. Roe and Schwartzman[72] also observed a greater increase in the total blood sugar content of diabetic subjects after ingestion of galactose than in that of normal people, and this was subsequently confirmed by Althausen and Wever.[4][5]

From studies of the degree of galactosuria, Harding and Van Nostrand[30] have shown that galactose is well utilized by the normal human body. These authors gave 50 grains of galactose by mouth and found a utilization of 97% in half of their subjects. The galactose utilization in our thyreotoxic patients, as estimated by the urinary galactose excretion test, has been 95% after the ingestion of 40 grammes of galactose. Harding and Van Nostrand[30] found in their patients pronounced variability in blood glucose changes after the administration of galactose.

Bianco[10] found no galactose in the peripheral blood of rabbits after the oral administration of 2·0 grammes of galactose.

Roe and Cowgill,[70] by simultaneously collecting samples of portal and hepatic blood from rabbits after the injection of galactose into the duodenum, were able to show significant withdrawals of galactose from, and corresponding additions of glucose to, the blood which passed through the liver; but they were unable to show any significant arterio-venous differences in galactose concentration in the blood of the leg or of the brain of the dog. Harding and Grant,[26] however, found well-defined arterio-venous differences in the total sugar content after ingestion of galactose; part of this was due to galactose and part to glucose, but as they could not eliminate the possibility of conversion of galactose to glucose, they did not conclude that there was necessarily direct galactose utilization by the tissues.

Wierzuchowski[80][84][85][86] reported that the assimilation of galactose by the dog was not affected by the type of feeding, by hunger or by the giving of adrenaline or thyroxin, and that insulin only slightly diminished the urinary output of galactose.

Cori (1926)[16] observed that in the rat the rate of formation of glycogen in the liver was much slower with a galactose diet than with glucose.

Cori (1925)[17] and Day[22] have shown that the rate of absorption of galactose is greater than that of any other sugar in the rat; but this appears to apply only to the rat.

Harding and Grant[25] and Harding and Van Nostrand[30] have concluded that the urinary threshold for galactose must be extremely low. In our patients 6·0 milligrammes *per centum* was the lowest peak of a blood galactose curve, and one of these patients excreted 1·2 grammes of galactose in the urine.

King and Aitken[50] reported that in their experience the blood galactose level after the oral administration of galactose might be anything from a few to 60 milligrammes *per centum* in the normal subject, and therefore they recommended that the oral galactose tolerance test should be replaced by the intravenous galactose tolerance test. King, Harrison and Delory[51] obtained elevated intravenous galactose tolerance curves in rabbits in which toxic hepatitis had been produced by carbon tetrachloride.

After the intake of a single dose of glucose, either orally or parenterally, man and laboratory animals are able to deal more rapidly with a subsequent dose of glucose; but Corley[18] found that after the administration of glucose or galactose the ability of the rabbit to deal with a subsequent dose of galactose did not increase.

Galactosuria as a Test of Liver Function.

For many years now reports have appeared of results obtained with a galactose tolerance test which depended on the degree of galactosuria produced by the oral ingestion of 40 grammes of galactose. According to Bauer,[3] not more than 3·0 grammes of galactose should be excreted in the urine in the following five hours. Some authors, including Lichtman,[56][57][58] and Shay and co-workers,[77][78][79][80] have spoken highly of this test, especially for the differential diagnosis of jaundice; but most writers condemn it because of the irregularity of

their results, and it has generally fallen into disfavour. De Lor and Reinhart,[34] after an extensive investigation of liver function tests, concluded that the galactose tolerance test, as estimated by the urinary excretion of galactose, was less reliable as a test of liver function than the hippuric acid excretion test, the bromsulphalein test, or estimation of the blood prothrombin or blood cholesterol levels.

In 1912 Hirose[22] reported that patients with thyreotoxicosis with this urinary galactose tolerance test gave results similar to those obtained from patients with cirrhosis of the liver and catarrhal jaundice; but Maclagan's results did not confirm this report, and he concluded that the urinary excretion of galactose depended too much on the rate of urinary flow to be of clinical use.

In our thyreotoxic patients we have found no clear relationship between the height of the blood galactose level, the degree of galactosuria, the rate of urinary flow, the results of other so-called liver function tests or the degree of thyreotoxicosis (Table II); and now, as a rule, we do not estimate the urinary galactose content when following the blood changes of a galactose tolerance test.

It is important to distinguish clearly between the galactose tolerance test, which depends on the degree of galactosuria and which was introduced in 1906 by Bauer,[42] and the galactose tolerance test of Althausen and co-workers,[1][2][4] in which the level of galactose in the blood is followed.

The Blood Galactose Tolerance Test in Thyreotoxicosis.

In an attempt to throw some light on the deranged carbohydrate metabolism of thyreotoxicosis, Althausen and Wever[4] investigated the changes in the blood glucose and galactose contents after the administration of 40 grammes of galactose in 400 millilitres of water. They found that while the average blood galactose curve of patients suffering from *diabetes mellitus* did not differ from that of normal subjects, the average blood galactose curve of patients with thyreotoxicosis increased in thirty minutes from zero to 52·8 milligrammes per 100 millilitres of blood. The average rise of galactose in the blood in thirty minutes in normal and diabetic subjects was only 15 milligrammes *per centum*. After thyreoidectomy the abnormal galactose tolerance curves of thyreotoxicosis were restored to normal.

One of the diabetic patients in Althausen and Wever's series gave a galactose tolerance curve rising to 77 milligrammes *per centum* in thirty minutes; but it seems possible from the consistently low figures obtained in diabetes since then by Althausen, Lockhart and Soley,[2] and by Maclagan and Rundle,[30][31] that this patient may also have been suffering from thyreotoxicosis.

Althausen and Wever[4] suggested that the high galactose tolerance curve of thyreotoxicosis was due either to accelerated intestinal absorption or to a reduced capacity to utilize galactose, probably due to hepatic damage. They pointed out that reduction in liver glycogen occurred in both thyreotoxicosis and diabetes, so that the difference in the galactose tolerance curves in those two conditions could not be due entirely to the low hepatic glycogen content. They also noticed that the changes in the galactose tolerance curve were often not proportionate to the severity of the thyreotoxicosis as judged by any one criterion; but because of the constancy of the so-called "impaired tolerance" they suggested that the test might be of value in the diagnosis of thyreotoxicosis.

Althausen,[1] and Althausen, Lockhart and Soley[2] have followed up the original work of Althausen and Wever[4] with the galactose test, and their results show that elevated blood galactose curves occur in thyreotoxicosis, diseases of the liver and *osteitis deformans*. In a group of 130 patients with thyreotoxicosis they obtained a positive response to the galactose test in 124 cases, a doubtful response in five and no response in one—that is, the test produced a positive result in 95·4%. They took 40 milligrammes of galactose per 100 millilitres of blood as the upper limit of normal. Althausen, Lockhart and Soley[2] obtained normal curves in anxiety states, in non-toxic diseases of the thyreoid, and in conditions other than thyreotoxicosis in which the basal metabolic rate may be elevated, and therefore they concluded that the galactose

TABLE II.

Galactose and Liver Function Tests on Thyreotoxic Patients.

Case Number.	Age of Patient in Years.	"Galactose Index."	Blood Galactose Peak. (Milligrammes *per centum*.)	Galactosuria. (Grammes.)	Urine Volume. (Millilitres.)	Basal Metabolic Rate. (Percentage.)	Prothrombin. (Percentage.)	Bilirubin. (Milligrammes *per centum*.)	Hippuric Acid Test. (Percentage.)
I	55	142	84	2·0	320	27	100	0·3	101
II	49	93	45	1·2	482	26	97	0·1	101
II[1]	49	48	32	1·6	326	5[5]	100	0·1	92
III	70	383	148	1·2	146	47	89	0·5	27
IV	23	62	36	0·8	307	43	100	0·1	—
V	19	11	9	0·6	275	22	89	0·5	122
VI	30	117	54	5·7	253	76	95	0·3	100
VII	40	65	56	5·3	280	54	94	0·9	103
VIII	34	249	115	3·9	267	66	—	0·1	101
VIII[1]	34	102	62	2·0	204	10[5]	100	—	104
IX	54	94	57	0·7	295	19	96	0·3	67
X	31	104	68	6·2	416	54	100	0·1	100
X[1]	31	54	42	1·5	585	15[5]	99	0·1	86
X[2]	31	80	30	4·7	620	20[5]	94	0·1	100
XI	37	190	70	4·2	186	56	100	0·3	100
XI[3]	37	230	105	4·4	638	25	77	—	93
XI[4]	37	200	82	6·7	398	32	98	—	90
XI[4]	37	57	36	8·0	425	35	100	0·1	—
XII	63	283	96	1·6	160	15	110	1·4	81
XIII	22	129	53	0·8	432	50	96	0·2	101
XIII[1]	22	93	49	0·5	75	7	—	—	—
XIV	59	300	121	3·5	282	66	100	0·1	89
XV	47	160	70	2·0	127	14	94	0·1	102
XVI	60	182	96	—	—	35	—	0·7	77
XVI[3]	60	129	74	4·5	255	22	—	0·4	80
XVIII	44	274	181	3·6	595	47	70	0·1	102
XX	52	151	56	2·4	488	52	100	0·4	106
XXI	51	220	105	2·8	545	62	84	0·3	92
XXVI	32	185	85	2·1	170	63	100	—	89
XXVII	49	129	74	0·8	—	15	—	—	—
XXVIII	36	96	47	1·0	—	25	—	—	97
XXIX	42	351	136	0·2	32	50	100	—	104
XXIX[1]	42	19	9	0·5	59	13[5]	100	—	—
XXX	26	315	123	3·0	219	43	100	—	99
XXX[1]	26	8	6	0·4	280	12[5]	100	—	—

[1] Results obtained after subtotal thyreoidectomy.
[2] Results obtained after a prolonged course of sedation and iodine therapy.
[3] Results obtained after a right hemi-thyreoidectomy and a course of deep X-ray therapy to the left lobe, but with persistence of thyreotoxicosis.
[4] Results obtained after a further course of deep X-ray therapy.
[5] The figure was below zero.

tolerance test might be of use in the diagnosis or exclusion of these conditions.

By taking blood samples five minutes after the ingestion of galactose, Althausen, Lockhart and Soley[2] found that galactose appeared in the blood much sooner in thyreotoxic patients than in normal subjects, and by intravenous galactose-tolerance tests they were able to show that the utilization of galactose by thyreotoxic patients was within normal limits. From these two observations they deduced that the high blood galactose levels in thyreotoxicosis were due to increased intestinal absorption. These authors were unable to determine a definite relationship between the basal metabolic rate and the results of the galactose tests, and they concluded that the intestinal absorption of sugar was more sensitive to the influence of a small excess of circulating thyroxin than was the basal metabolic rate, but that when the basal metabolic rate was found to be elevated in patients with thyreotoxicosis, then the basal metabolic rate offered a more accurate index of the degree of thyreotoxicosis than did the galactose test.

To complicate matters, Althausen, Lockhart and Soley[2] found elevated blood galactose curves in single cases of *diabetes mellitus*, congenital syphilis with chronic mastitis, hyperinsulinism, intractable gastric ulcer, intestinal hypermotility, Cushing's disease, and prolonged fever of unknown origin; but the possibility of the coexistence of hyperthyreoidism, hepatitis or *osteitis deformans* does not appear to have been eliminated in these cases.

The association of jaundice with thyreotoxicosis was first pointed out by Dring[129] in 1874, and later by Eger[129] and Eder;[127] but within recent years increasing prominence has been given by Bartels,[6] Bartels and Perkin,[7] Beaver and Pemberton,[9] Boyce and McFetridge,[11,12,13] Cameron and Karunaratne,[14] Elmer,[29] Haines, Magath and Power,[34] Lahey,[52] Lord and Andrus,[59] Shaffer,[79] Weller,[92] and Wyndham[93] (to mention only a few of the authors concerned) to the liver changes and impaired liver function which so frequently accompany thyreotoxicosis. Similar liver changes have been found by Hashimoto[37] in rats fed on toxic amounts of thyreoid. Kerr and Rusk[49] have reported a case of thyreotoxicosis in which the liver changes resembled those of acute yellow atrophy, and a similar case has been reported by Wyndham.[93]

After the administration of thyroxin to a group of five rabbits, Maclagan and Rundle[61] found impaired utilization of galactose as shown by intravenous galactose-tolerance tests. They also found degenerative lesions in the livers of these rabbits. The results of the intravenous tests are at a variance with those of Althausen, Lockhart and Soley[2] for thyreotoxic patients. King and Aitken[50] have reported the finding of a normal intravenous blood galactose curve in myxœdema. These series of intravenous tests of Althausen, Lockhart and Soley,[2] and of Maclagan and Rundle,[61] are both too small if significant deductions are to be made from them. The results of many more intravenous galactose tolerance tests require investigation before the existence of impaired utilization of galactose in thyreotoxicosis can be deduced from these tests.

Maclagan[60] has suggested that there may be a difference in the ability of the liver to deal with galactose arriving by the portal vein or by the general circulation. This may be; but it is a matter rather difficult of proof. A similar objection had previously been raised by Maclean and de Wesselow[62] to intravenous glucose tolerance tests.

In England, Maclagan and Rundle[61] have reported results with the galactose tolerance test, but they interpreted the high curves of thyreotoxicosis as evidence of impaired liver function.

Maclagan and Rundle[61] carried out galactose tolerance tests on 41 thyreotoxic patients and reported that 30 of these patients showed impairment of glycogenic function of a degree comparable with that seen in toxic jaundice. They found that although the basal metabolic rate decreased in these patients with the administration of sedatives and iodine, the galactose tolerance curve remained elevated before operation. Within two weeks of operation the curve usually returned to normal. This fact, that the galactose tolerance test is relatively unaffected by sedation and iodine therapy, suggests the occasional use of this test for diagnosis in cases in which the patient's condition has been so improved by the medical treatment given by the local practitioner that the pulse and basal metabolic rates have both returned to normal and the thyreotoxic symptoms practically disappeared before his admission to hospital.

Discussion.

At Prince Henry Hospital the technique of Maclagan has been followed. Forty grammes of galactose are given in 300 millilitres of water and the blood galactose level is estimated at intervals of half an hour for the next two hours. Maclagan[60] has applied the term "galactose index" to the sum of these four values in milligrammes of galactose per 100 millilitres of blood, and he found that the mean galactose index for 50 normal people was 68, with a standard deviation of 39·3 and with 130 as the upper normal limit. At the Prince Henry Hospital we have adopted his terminology and normal limits.

Ross and Tonks[74] have succinctly expressed in the following words their reason for using a fixed dose of glucose in glucose tolerance tests: "There can be little reason to think that equal weights of thin and obese, well and ill, normal and endocrinely disordered, active and inactive patients will be metabolically equivalent."

A review of the results obtained by previous observers with fixed quantities of galactose and with quantities of galactose dependent on body weight, reveals similar variation in each series. Because of this, and because Ross and Tonks's argument seems equally applicable to tolerance tests other than those of glucose, we have used a fixed dose of galactose for our patients.

In an attempt to analyse Maclagan and Rundle's assumption[61] that the high galactose tolerance curve of thyreotoxicosis is due to impaired liver function, hippuric acid excretion tests (Quick[85]) and prothrombin and bilirubin estimations have been carried out on the thyreotoxic patients in this series on whom galactose tests were performed (Table II). Although the total number of galactose tests is small (62), it is apparent from our results that the galactose tolerance curve may be elevated while three of the functions of the liver, as estimated by these tests, are within normal limits. None of these patients had a macrocytic anæmia which might have been attributed to hepatic disease. In those thyreotoxic patients (Cases III, IX, XII and XVI) who showed interference with the excretion of hippuric acid, it was impossible to correlate the hippuric acid excretion with the height or the shape of the galactose tolerance curve. Again, some patients with impaired liver function (Cases XXXV and L), as estimated by these tests and due to lesions of the biliary tract, have shown normal galactose tolerance curves.

While it cannot be argued from normal results with the hippuric acid excretion test and prothrombin and bilirubin estimations that all the liver functions are necessarily normal, nevertheless it is interesting that it should be possible to obtain these normal readings while the galactose tolerance curves are abnormal, if, as Maclagan and Rundle[61] suggest, the high galactose tolerance curves are indicative of impaired liver function.

Quick[85] pointed out the necessity for determining, as far as possible, that the renal function was normal before the hippuric acid excretion test was utilized as a test of hepatic function. In none of the cases discussed in this paper has the examination of the urine revealed any abnormality; and the blood urea level, which was estimated in most of the cases, was always within normal limits.

Boyce and McFetridge[11,12,13] reported impairment of liver function, as shown by the hippuric acid excretion test, in all patients, and especially in thyreotoxic patients, in the days following general or spinal anæsthetics. Similarly, Bartels[6] has demonstrated that on the sixth or seventh day after operation there is impairment of liver function. Althausen, Lockhart and Soley,[2] and Maclagan and Rundle[61] have shown that after thyreoidectomy there is a rapid return of the galactose tolerance curve to normal, and this has been our experience.

TABLE III.

Galactose and Liver Function Tests on Patients with Diseases other than Thyreotoxicosis.

Case Number.	Age of Patient in Years.	"Galactose Index."	Blood Galactose Peak. (Milligrammes *per centum*.)	Galactosuria. (Grammes.)	Urine Volume. (Millilitres.)	Basal Metabolic Rate. (Percentage.)	Prothrombin. (Percentage.)	Bilirubin. (Milligrammes *per centum*.)	Hippuric Acid Test. (Percentage.)	Diagnosis.
XXXI	36	40	17	0·1	87	26	88	—	85	Chronic myeloid leuchæmia.
XXXII	21	120	66	3·0	298	7[4]	—	—	—	Anxiety neurosis.
XXXIII	20	18	9	0·3	552	3[4]	100	0·5	104	Colloid goitre.
XXXIV	42	83	39	2·2	93	—	75	5·5	48	Stricture of common bile duct.
XXXV	32	82	35	0·6	230	—	—	—	105	Pulmonary tuberculosis.
XXXVI	40	84	30	0·5	110	—	—	2·3	95	Hæmochromatosis.
XXXVII	67	30	15	Nil	640	26[4]	100	0·3	87	Myxœdema.
XXXVII[3]	67	49	30	2·0	523	18	100	0·3	90	Myxœdema.
XXXVIII	55	242	92	3·6	360	1[4]	100	0·3	96	? Thyreotoxicosis.
XXXVIII	55	212	105	7·3	488	13[4]	—	—	—	? Thyreotoxicosis.
XXXIX	25	23	19	4·6	295	15	100	0·4	87	*Diabetes mellitus*.
XL	30	56	39	1·4	535	15[4]	98	0·2	104	Non-toxic adenoma of thyreoid.
XL[1]	30	69	39	2·9	382	3[4]	100	0·1	100	Non-toxic adenoma of thyreoid.
XLI	28	6	6	1·2	498	8	100	0·1	95	Non-toxic adenoma of thyreoid.
XLII	38	91	39	5·6	560	6	100	0·1	106	Non-toxic adenoma of thyreoid.
XLIII	28	38	21	1·5	615	2	—	0·4	—	Anxiety neurosis.
XLIV	39	30	18	2·6	300	4	94	0·5	72	Non-toxic adenoma of thyreoid.
XLV	70	38	19	Nil	566	—	73	0·3	93	Subacute hepatitis.
XLVI	27	99	41	1·1	307	15[4]	—	—	105	Anxiety neurosis.
XLVII	62	255	83	4·2	190	—	95	0·5	103	Cirrhosis of liver.
XLVII[3]	62	282	88	0·4	85	—	72	0·3	37	Cirrhosis of liver.
XLVIII	56	170	46	1·2	48	—	75	5·9	50	Carcinoma of liver.
XLIX	38	36	21	0·2	498	10[4]	—	—	—	Anxiety neurosis.
L	64	Nil	Nil	0·3	44	—	—	4·4	—	Cholelithiasis pylorospasm.
LI	28	37	27	1·5	230	3	—	0·1	100	Non-toxic adenoma of thyreoid.

[1] Results obtained after subtotal thyreoidectomy. [2] Results obtained after treatment with thyroxin. [3] Results obtained after laparotomy.
[4] The figure was below zero.

In Case XLVII (cirrhosis of the liver) an abnormal galactose tolerance curve was found. Further elevation of the curve occurred after laparotomy, while the result of the hippuric acid excretion test decreased from 103% to 37%, owing, as would be expected in this case, to aggravation of the gross liver damage already present.

It would seem unlikely that in the week following thyreoidectomy the liver, which in all severely thyreotoxic patients presents pronounced degenerative lesions, should suddenly resume its normal capacity to deal with glucose and galactose, while its ability to conjugate benzoic acid with glycine is further impaired. Rather it is probable that the activity of the enzymes of the intestinal mucosa, when freed from the stimulating effect of the excess thyreoid hormone, would be lessened, and thus the rate of intestinal absorption would be decreased. It is easy to demonstrate after operation that there is a decrease of the accelerated peristaltic rate of thyreotoxicosis.

If, as Maclagan and Rundle[61] suggest, the impairment of liver function was the sole basis of the altered galactose metabolism, then a decreased rate of utilization of galactose after intravenous injection would have been expected in thyreotoxic patients; but Althausen, Lockhart and Soley[2] have shown the galactose utilization to be normal in a small series of such patients. Reference has already been made to the utilization of galactose in the body as a whole following oral ingestion. From the figures given this is of the same order in thyreotoxic patients as in normal subjects.

Since rapid utilization of glucose by the tissues occurs in thyreotoxicosis (a fact originally established by Sanger and Hun[75]) the typical elevated glucose tolerance curve of this disease is probably related more to the rapidity of absorption from the bowel than to impairment of glycogen function of the liver.

Eventually it may possibly be shown that both factors—the accelerated intestinal absorption and the impaired liver storage—play a part in thyreotoxicosis in the ætiology of the abnormal galactose metabolism.

Maclagan and Rundle[61] did not attempt to explain the low oral galactose tolerance curves obtained in myxœdema. According to their theory, these curves would be due to rapid removal of galactose by the liver; but decreased phosphorylation and intestinal hypomotility are more probable causes of the low blood galactose levels in myxœdema than a supernormal increase in the glycogenic function of the liver.

Maclagan and Rundle[61] suggested that in those patients who showed slight elevation of the galactose curves persisting for many months after operation, residual liver damage was present. It is necessary, however, to prove that sufficient thyreoid had been removed from these patients and that there still did not exist minor grades of thyreotoxicosis. In such cases it would be wise to have several estimations made of the basal metabolic rate and to check the pulse rate and clinical condition under strict hospital control.

Taking their patients in groups according to severity of their symptoms, these authors found a clear relationship between the clinical grade of the thyreotoxicosis, the basal metabolic rate and the galactose index. This relationship has been close in most of our cases at the Prince Henry Hospital; but we have also noticed, as did Maclagan and Rundle,[61] that in a few cases the galactose index has differed considerably from the value expected on the clinical findings and the basal metabolic rate.

Maclagan and Rundle's conclusion[61] that the clinical value of the galactose tolerance test is not necessarily affected by speculations as to its mechanism is important, and from the results obtained with this test at the Prince Henry Hospital this year we concur with this conclusion.

Sixty-two galactose tolerance tests have been carried out at the Prince Henry Hospital in the past year. Thirty-seven of these tests were on patients suffering from thyreotoxicosis. In one of these cases (Case VI) three galactose tests were performed at intervals of one week, since the anomalous result of no galactose appearing in the blood in the two hours following the ingestion of 40 grammes of galactose was twice elicited. The third test, however, gave a galactose tolerance curve more or less commensurate with the patient's degree of thyreotoxicosis, and it was then discovered on further questioning that she had eaten some fruit before each of the first two tests. By the administration of a glucose and galactose mixture it is possible, if both the blood glucose and the blood galactose levels are followed for the next four hours, to show that in some people the absorption of galactose from the bowel is inhibited until most of the glucose has been absorbed. In one other case (Case L) a similar absence of galactose from the blood in the two hours of the galactose test occurred; but it was found that in this case there was a delay in the emptying of the stomach. These two possible fallacies should be borne in mind. If there is any vomiting, this may of course interfere with the results of the test.

Nephritis may delay the disappearance from the blood of glucose and other carbohydrates in excess of the fasting

levels. Because of this, and because of the interference by impaired renal function with the hippuric acid excretion test, I have been careful to exclude as far as possible any patients with functional renal impairment.

The composite galactose curve derived from Maclagan and Rundle's[63] tests of 50 normal people is shown in Figure VIII. I have taken this as the average normal curve, and superimposed it on the other galactose graphs in this paper.

The typical blood galactose curve of thyreotoxicosis, rising rapidly to a peak above 40 milligrammes *per centum* and then returning nearly to normal within two hours, is seen in Figure IX. This curve was obtained from a female, aged twenty-six years, with a hyperplastic thyreoid gland, who had pronounced thyreotoxic symptoms

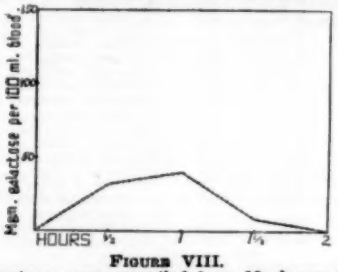

FIGURE VIII.
Galactose curve compiled from Maclagan and Rundle's results[63] in 50 normal subjects.

for six months. Four days before the galactose tolerance test her basal metabolic rate was +43% and her liver function, as estimated by the hippuric acid excretion test, was 99%. Four days after the galactose test a subtotal thyreoidectomy was performed. The galactose tolerance curve obtained thirteen days after this operation and when her basal metabolic rate had fallen to -12% is also shown in Figure VIII. Similar pre-operative and post-operative galactose curves have been obtained in other frank cases of thyreotoxicosis.

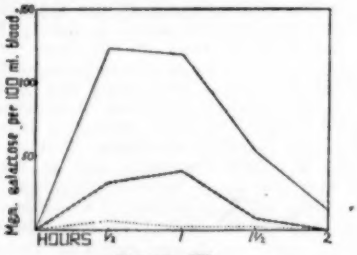

FIGURE IX.
Solid line: blood galactose curve from Case XXX (thyreotoxicosis) prior to operation. Dotted line: blood galactose curve from the same case two weeks after subtotal thyreoidectomy. Line with crosses: average normal galactose curve.

Case XII demonstrates the application of the galactose test to the diagnosis of masked thyreotoxicosis.

This patient, a male, aged sixty-three years, had suffered from cardiac failure for many years, during which time he had often required treatment in hospital. On his final visit some retrosternal dulness was noticed, the thyreoid was found to be slightly enlarged on palpation, and his basal metabolic rate was +15%. However, the galactose curve was elevated (Figure X) and confirmed the diagnosis of thyreotoxicosis; but unfortunately it was impossible to prevent further deterioration in his general condition, and death occurred without operation being feasible.

Apart from the diagnosis of masked thyreotoxicosis, the other important use of the galactose test is in the exclusion of thyreotoxicosis when the estimation of the basal metabolic rate is not possible or is not reliable.

In addition to an adenomatous thyreoid, the patient in Case XXV had "open" pulmonary tuberculosis. While pulmonary tuberculosis is exceptionally rare as a complication of thyreotoxicosis, it was necessary to determine whether her symptoms of loss of weight, sweating, nervousness, tachycardia, palpitation, weakness and dyspnœa were entirely due to tuberculosis or whether there was a superadded thyreotoxic element. Since tuberculosis may cause an increase in the basal metabolic rate, and since it was undesirable to contaminate the basal metabolism apparatus with tubercle bacilli, a galactose tolerance test was performed instead. When it was discovered that the galactose tolerance curve was within normal limits (Figure XI), it was apparent that the adenomatous thyreoid was non-toxic and that the symptoms were due to the pulmonary lesion.

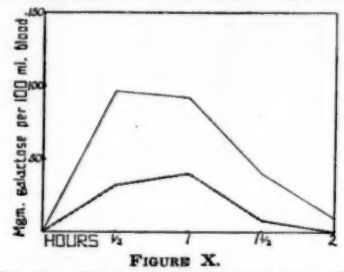

FIGURE X.
Solid line: blood galactose curve from Case XII, masked thyreotoxicosis with cardiac failure. Line with crosses: average normal galactose curve.

In myeloid leuchæmia associated with an elevated basal metabolic rate and the symptoms of weakness, dyspnœa, palpitation and loss of weight, there may be a superficial resemblance to thyreotoxicosis, especially if there is an associated non-toxic adenomatous enlargement of the thyreoid. The galactose tolerance curve in such a case[1] is illustrated in Figure XII.

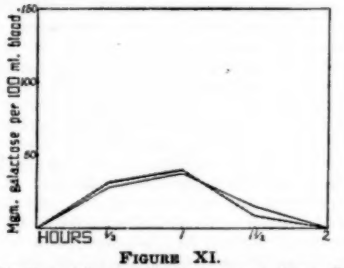

FIGURE XI.
Solid line: blood galactose curve from Case XXXV, non-toxic adenomatous thyreoid and pulmonary tuberculosis. Line with crosses: average normal galactose curve.

This patient, a female, aged thirty-six years, had complained of these symptoms for six months, but presented no tremor, exophthalmos or nervousness, although her thyreoid gland was irregularly enlarged and the basal metabolic rate was +26%. On clinical examination the spleen, liver and

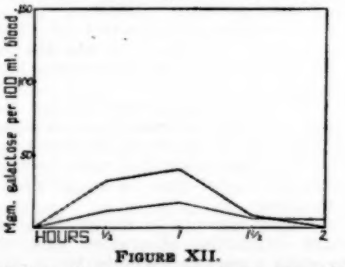

FIGURE XII.
Solid line: blood galactose curve from Case XXXI, non-toxic adenomatous thyreoid and chronic myeloid leuchæmia. Line with crosses: average normal galactose curve.

lymph glands were not enlarged, but it was noticed that instead of the high pulse pressure of thyreotoxicosis, her blood pressure was 88 millimetres of mercury systolic and 58 diastolic. There was slight pallor of the mucous membranes and a blood examination gave the following informa-

[1] This case was discussed at a clinico-pathological conference at the Prince Henry Hospital on May 26, 1941.

tion: the hæmoglobin value was 7·7 grammes *per centum*; the erythrocytes numbered 3,850,000 per cubic millimetre, 2·4% being reticulocytes; the platelets numbered 448,000 per cubic millimetre. The leucocytes numbered 230,000 per cubic millimetre; 49% were neutrophile polymorphonuclear cells, 2% were eosinophile cells, 1% were basophile cells, 26% were neutrophile metamyelocytes, 12% were myelocytes, 4% were premyelocytes, 2% were myeloblasts and 4% were lymphocytes. The bone marrow obtained by sternal puncture was also diagnostic of chronic myeloid leuchæmia.

While it is often difficult correctly to estimate the basal metabolic rate of patients with anxiety neurosis and associated hyperventilation, the problem of the differential diagnosis of this condition from thyreotoxicosis is simplified by the galactose test. Figure XIII shows the galactose tolerance curve from a patient suffering from an anxiety neurosis. With sedation and rest her hyperventilation disappeared and the basal metabolic rate decreased to −10%.

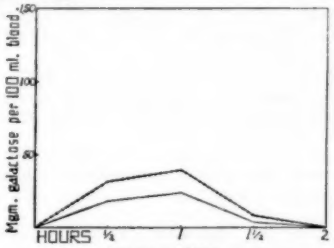

FIGURE XIII.
Solid line: blood galactose curve from Case XLIX, anxiety neurosis. Line with crosses: average normal galactose curve.

In Figure XIV is shown the galactose tolerance curve from a patient, aged forty-seven years, thought to be suffering from an anxiety neurosis. Her basal metabolic rate varied from +14% to −14%, and her thyreoid was slightly enlarged. In view of the elevated galactose tolerance curve, and incidentally a high glucose tolerance curve, while the result of the hippuric acid excretion test and the prothrombin and bilirubin levels were normal, thyreoidectomy was performed, with subsequent relief of symptoms and restoration of the galactose tolerance curve to normal.

Mention has already been made of the normal galactose tolerance curves obtained in diabetes by Althausen, Lockhart and Soley[29] and by Maclagan and co-workers.[30][31]

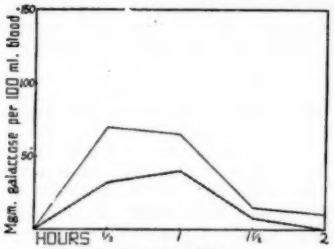

FIGURE XIV.
Solid line: blood galactose curve from Case XV, possible thyreotoxicosis. Line with crosses: average normal galactose curve.

In Case XXXIX frank diabetes had been present for nine months when an adenomatous enlargement of the patient's thyreoid was noticed. For the previous two years there had been symptoms suggestive of mild thyreotoxicosis. With the administration of sedatives and iodine the basal metabolic rate decreased from +15% to −7%. The patient's galactose tolerance curve on her admission to hospital was within normal limits (Figure XV); thus the thyreotoxic element, if any, must have been very slight. Because of difficulty in controlling her diabetic condition a subtotal thyreoidectomy was performed. After this her resistance to insulin decreased.

Herbert and Davison[33] have reported elevated blood lævulose curves in two cases of hæmochromatosis. It was with interest that a galactose test was performed in a proved case of hæmochromatosis in which the glucose tolerance curve was of the diabetic type. Opportunity had been afforded at operation last year for a biopsy of the patient's liver. Dr. F. B. Byrom's report on the sections confirmed the diagnosis. This patient's liver function, as estimated by the hippuric acid excretion test, was 95% of normal, and his galactose tolerance curve (Figure XVI) approximated to normal, except that it had not returned to zero within two hours.

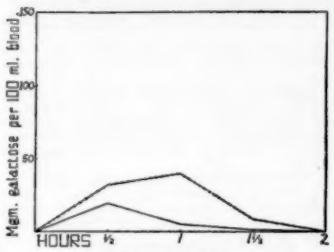

FIGURE XV.
Solid line: blood galactose curve from Case XXXIX, *diabetes mellitus* with mild thyreotoxicosis. Line with crosses: average normal galactose curve.

The reason for the reproduction of this and the next graph (Figure XVII) is that although there were gross histological changes in the livers of these patients, they both presented galactose tolerance curves within normal limits. Only three of the thyreotoxic patients in this series have come to autopsy, two without operation and one after operation (Cases III, XXI, and IX); but in each case

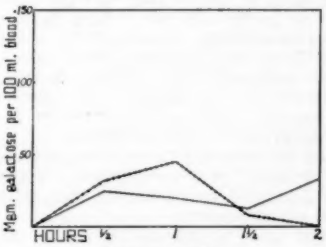

FIGURE XVI.
Solid line: blood galactose curve from Case XXXVI, hæmochromatosis. Line with crosses: average normal galactose curve.

pronounced degenerative lesions were present in the liver. In two other cases in which galactose tests have been carried out, it has been possible to examine the liver. The final diagnosis in these two cases was (i) subacute hepatitis due to cholangitis and (ii) cirrhosis of the liver.

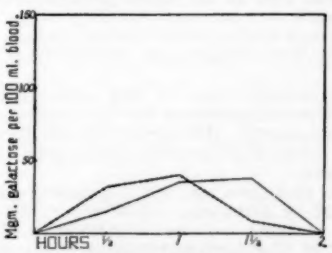

FIGURE XVII.
Solid line: blood galactose curve from Case XXXIV, stricture of the common bile duct. Line with crosses: average normal galactose curve.

Thus on seven occasions microscopic examination of the liver has been performed in cases in which galactose tests have also been carried out. In these few cases it has not been possible to correlate the histological changes with the results of the galactose tolerance test.

Figure XVII shows the galactose tolerance curve from a patient with severe liver damage secondary to a stricture of the common bile duct. Figure XVIII shows the galactose tolerance curve from a patient with cirrhosis of the liver and with apparently a similar degree of liver damage to the patient from whom Figure XVII was obtained. The difference in the two curves is striking and it indicates the difficulty in attempting to explain variations in the galactose tolerance curves.

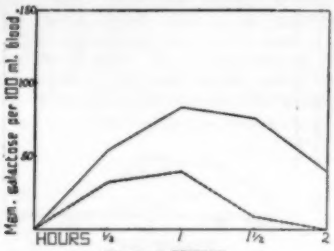

FIGURE XVIII.

Solid line: blood galactose curve from Case XLVII, cirrhosis of the liver. Line with crosses: average normal galactose curve.

Summary.

Many of the more important papers dealing with carbohydrate metabolism in thyreotoxicosis are reviewed.

Test meals and opaque meal examinations have been performed in a small series of thyreotoxic patients. The frequent occurrence of achlorhydria in thyreotoxicosis has been confirmed (in all of a series of eight), but it was also found in each of these cases that the stomach emptied within forty or sixty minutes. This rapidity of gastric emptying, and in addition, hypermotility of the bowel, were demonstrated in a small series of thyreotoxic patients by opaque meal examinations. After thyreoidectomy the accelerated peristalsis is retarded. It is suggested that this rapidity of gastric emptying is a factor in accelerating intestinal absorption in thyreotoxicosis.

It is pointed out that the terms "impaired tolerance" and "increased tolerance" are insufficiently comprehensive to apply to the deranged carbohydrate metabolism of thyreotoxicosis and of myxœdema.

The common types of glucose tolerance curves found in thyreotoxicosis are shown, but no one curve can be said to be diagnostic of thyreotoxicosis. These curves tend to return to normal after thyreoidectomy.

The "flat" glucose tolerance curve of myxœdema is shown, as are also the glucose tolerance curves of a myxœdematous patient after the exhibition of thyreoid extract and after the exhibition of thyroxin.

The renal threshold for glucose of the thyreotoxic patients in this series was elevated, and it is suggested that this was due to increased phosphorylation in the renal tubules.

The literature dealing with the metabolism of galactose and with galactosuria as a test of liver function has been reviewed.

In the thyreotoxic cases in this series there has been no clear relationship between the blood galactose level, the degree of galactosuria, the rate of urinary flow, the results of other so-called liver function tests, and the degree of thyreotoxicosis.

The blood galactose test for thyreotoxicosis was introduced in 1937 by Althausen and Wever.[40] The results and conclusions of these authors, of Althausen, Lockhart and Soley,[39] and of Maclagan and Rundle,[41] are discussed.

Sixty-two blood galactose tests have been carried out at the Prince Henry Hospital in the past year, and 37 of them applied to patients suffering from thyreotoxicosis. In nearly all cases hippuric acid excretion tests and prothrombin and bilirubin estimations were also performed. From these results and for other reasons given it is concluded that while Althausen, Lockhart and Soley's theory is that the abnormal galactose curves of thyreotoxicosis are due to accelerated intestinal absorption, and Maclagan and Rundle's theory is that the abnormal curves are solely dependent on impaired liver function, probably the correct explanation is a combination of the two theories—that is, that the derangement of galactose metabolism in thyreotoxicosis consists chiefly in accelerated intestinal absorption and to a lesser extent in impairment of the glycogenic function of the liver.

The possible uses of the galactose tolerance test are indicated—in masked thyreotoxicosis, in thyreotoxicosis after medical treatment, and in cases in which the basal metabolic rate is not available, is not reliable, or is affected by pathological conditions other than thyreotoxicosis.

In some cases there has been a great discrepancy between the "galactose index" and the value expected from the clinical findings and the basal metabolic rate. Such cases are being further investigated.

Conclusions.

Intestinal hypermotility occurs with thyreotoxicosis, but decreases again within a week after thyreoidectomy. This hypermotility is probably a factor in accelerating intestinal absorption in thyreotoxicosis.

Achlorhydria is a common finding in thyreotoxicosis.

The fasting blood sugar level of 23 thyreotoxic patients was 108 milligrammes *per centum*. None of these patients showed glycosuria while fasting.

The delayed fall in the blood sugar curve from its peak found in some thyreotoxic patients probably represents impaired glycogenic function of the liver and not incipient diabetes.

In addition to the increased intestinal phosphorylation, it is suggested that there is also an increase in the phosphorylation of the renal tubules in thyreotoxicosis; the renal threshold for glucose is thus raised and the degree of galactosuria decreased.

The galactose utilization in thyreotoxic patients was of the same order as in normal subjects.

Galactose absorption is delayed when there is interference with the emptying of the stomach and when the bowel contains glucose in addition to the galactose.

The probable absence of a renal threshold for galactose is confirmed.

The derangement of galactose metabolism in thyreotoxicosis consists chiefly in accelerated intestinal absorption and to a less extent in impairment of glycogenic function of the liver.

While the blood galactose test is of importance clinically for the diagnosis or exclusion of thyreotoxicosis, further study of the results of this test are necessary in order that its limitations may be fully understood.

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ARTERIAL HYPERTENSION: A SYMPTOM OF INTRACRANIAL TUMOURS.

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ANOMALIES of arterial pressure as a symptom of intracranial tumour are not mentioned in most text-books of neurology and internal medicine. The relation between anomalies of arterial pressure and intracranial tumours has been discussed for the first time in a painstaking manner by Upmark in his monograph "The Carotid Sinus and the Cerebral Circulation" (1935). Upmark's material was represented by 486 patients with verified intracranial tumours operated upon by Dr. Cushing. The ages of the patients varied from twenty to forty-four years. In 20% the systolic blood pressure was low, less than 100 millimetres of mercury; in only 4% the diastolic blood pressure was high, more than 100 millimetres of mercury. Upmark noted that high blood pressure was far more commonly associated with infratentorial than with supratentorial tumours.

In a recently published paper[1] five cases of brain tumour localized in the posterior fossa have been described, in which arterial hypertension was present. The patients were respectively aged fifty-two, fifty-five, fifty-six, fifty-eight and sixty-six years. The diagnosis of brain tumour was made either at necropsy or shortly before death. Not one of the five patients was successfully operated on, and therefore no proof was given that the arterial hypertension was due to the presence of the brain tumour.

Reports of Cases.

The two cases now to be described illustrate brain tumours associated with high degrees of arterial hypertension; the first lesion was a meningioma in the posterior fossa, the second an astrocytoma in the supratentorial space. In both cases the return to normal blood pressure has been observed after the successful complete removal of the tumour.

CASE I.—B.A., aged forty-three years, had always been in good health till two years ago, when he suffered from vomiting every morning for three months. He continued to work at bricklaying till six months ago, when headache and dizziness appeared. The patient's wife said that the symptoms came on suddenly, and were accompanied at first by nausea, retching and fainting. She said that the headache and giddiness had not progressed in severity; the patient considered that they had done so. The headache came in attacks, starting from the neck and radiating upwards onto the skull, and then forward over the right side of the head and face. During the attacks the patient felt that the face was congested, and there was a sensation of numbness or of "pins and needles" in the right cheek.

Examination revealed a pale-complexioned man of asthenic habitus. The internal organs were normal. The pulse rate was 74 per minute; the blood pressure was permanently high, 170 to 180 millimetres of mercury systolic and 110 millimetres of mercury diastolic. Chemically and microscopically the urine was normal and its specific gravity was 1,020. The patient was mentally bright; he ate and slept well.

In the fairly large skull, the horizontal circumference of which measured 57 centimetres, no abnormality was discovered on palpation, percussion or auscultation. Both eyes had a central vision of $^6/_6$; the visual fields were normal, except for a slight enlargement of the blind spot. The ophthalmoscopic examination revealed bilateral multiple retinal hæmorrhages, situated partly on the border of the disk, partly along the larger vessels. Some hypæsthesia for cotton wool and hypalgesia for pin pricks on the right side of the face, head and neck were noted. The right lower facial musculature was less innervated than the left, and the right nasolabial fold was absent. A considerable horizontal nystagmus to the right side, less to the left side, was present, along with past-pointing with the right hand laterally and downward. Romberg's sign was present. The right limbs were hypotonic; pendulum-like excursions of the right leg accompanied the knee jerk.

An X-ray examination of the skull revealed a symmetrical hyperostosis of the vault, thickest in the parietal region, where it measured 16 millimetres. The structure of the cranium was spongy, with smaller and larger islands of sclerosis in the parietal and occipital bones resembling Paget's disease. There were no signs of increased intracranial pressure, nor was any calcification seen inside the cranial cavity. The base of the skull had a normal appearance. The antero-posterior view in Towne's position disclosed abnormally wide internal auditory canals; each measured 8·0 millimetres, the normal measurement being 5·0 millimetres.

During the ensuing two months the patient's symptoms did not show much alteration; attacks of headache, the unsteady gait, permanent arterial hypertension, numbness of the right side of the face, slight right-sided facial weakness and nystagmus continued. The ophthalmoscopic findings revealed no pronounced changes. The symptoms suggested a right-sided cerebellar process, either a vascular condition, most likely a partial thrombosis of the posterior inferior cerebellar artery, due to essential hypertension, or a neoplasm in the right cerebellar fossa, causing arterial hypertension or accidentally combined with essential hypertension.

At this time the pressure of the lumbar cerebro-spinal fluid, of which the protein content was 100 milligrammes per 100 cubic centimetres, was 210 millimetres of water. As the next diagnostic step, a bilateral ventricular estimation was performed, by means of the posterior parietal approach. During this procedure, the bone of the right posterior parietal region was found to be sclerosed; on the left side it was soft and vascular. Both lateral ventricles were estimated to be equally enlarged and the fluid therein to be at an abnormally high pressure. Suboccipital craniotomy

was undertaken immediately after the ventricular estimation (December 9, 1940). The squamous portion of the occipital bone showed a very dense structure on the right side, whereas the structure on the left side was spongy. A sharply circumscribed tumour, attached to the wall of the right lateral sinus, was found under the dura covering the right cerebellar lobe. The tumour, a typical meningioma, was radically removed from the neighbouring structures. The convalescence was uneventful; the blood pressure dropped to normal levels (138 millimetres of mercury systolic and 95 diastolic) after the operation and remained low for the following months.

CASE II.—J.G., aged nineteen years, was a farm labourer. The patient's father and mother died suddenly from cardiac disease, each at the age of fifty-four years. The mother's death was preceded by high blood pressure. The patient was well until the age of eighteen years, when he complained of headache and a sensation of blood rushing to the head and ears. Some weeks later he began to suffer from sick turns with vomiting, and was aware that he could not see to his right side. Objects seen with the left eye sometimes seemed to have a dark colour. As the patient had a high blood pressure the diagnosis of essential hypertension was made and the patient was treated by his local doctor in his home town with a special diet.

When the patient entered hospital his blood pressure measured constantly 170 millimetres of mercury systolic and 100 diastolic. The internal organs and the urine were normal. Right homonymous hemianopia was present, together with bilateral papillœdema, which was more pronounced in the left eye. Visual acuity was $^6/_{24}$ on the left and $^6/_6$ on the right. A lumbar puncture was made; the cerebro-spinal fluid, which contained 50 milligrammes of protein per 100 cubic centimetres, had a pressure of 500 millimetres of water.

X-ray examination of the skull revealed digitations and widening of the sutures and enlargement of the *sella turcica*, indicating increased intracranial pressure. Two areas of calcification were visible in the left occipital region just above the tentorium. One of the calcifications was the size of a bean and was dense and sharply outlined; the other cast a faint ill-defined shadow.

The ophthalmological and radiological findings suggested a tumour in the left occipital lobe. Operation revealed a large tumour in the left occipital lobe. Although the margins of the tumour were not sharply defined, it was possible to remove it by suction from the adjacent part of the brain. Microscopic examination showed the tumour to be an astrocytoma. When the patient left hospital five weeks later his blood pressure was low; in three months' time the systolic blood pressure was 120 millimetres of mercury.

Comment.

In the first case there was a difficulty in the differential diagnosis between a cerebellar syndrome due to vascular disturbances, produced by essential hypertension, and a tumour of the posterior fossa causing arterial hypertension. The history and the development of the clinical symptoms, a right-sided cerebellar syndrome, swelling of the disk and retinal hæmorrhage, and a persistent systolic blood pressure of 170 millimetres of mercury, did not allow a sure diagnosis. The X-ray findings in the skull, a diffuse cranial hyperostosis giving the appearance of Paget's disease, and enlargement of both internal acoustic canals, were not helpful; the hyperostosis could be interpreted as an occasional coincidence or as a meningioma hyperostosis. The widening of the internal auditory canals could be explained as a normal variation, especially as it was so symmetrical. Of diagnostic importance were the high level of protein in the cerebro-spinal fluid and the result of the ventricular estimation; both lateral ventricles were enlarged and the ventricular pressure was high.

The operation, a bilateral suboccipital craniotomy, revealed a considerable sclerosis of the occipital squama on the right side, whereas on the left side the structure of the bone was spongy. A large tumour, a meningioma of the right posterior fossa, was found and radically removed. The findings in the occipital squama may give an explanation of the character of the diffuse cranial hyperostosis with partial sclerosis; it was probably a meningioma hyperostosis.

After the operation the patient recovered quickly and completely. The blood pressure returned to normal levels.

The case is interesting from different standpoints:

1. A continuously raised arterial blood pressure due to the presence of a cerebellar meningioma dropped to normal levels after the successful removal of the tumour.

2. There were difficulties with regard to the differential diagnosis between a tumour of the cerebellar fossa and a partial thrombosis of the posterior inferior cerebellar artery due to essential hypertension.

3. There were difficulties in the differential diagnosis between papillœdema and hypertensive neuro-retinitis.

4. An unusual type of diffuse cranial hyperostosis with partial sclerosis of the diploë was present, most probably a meningioma hyperostosis.

5. The widening of both internal auditory canals represented the only radiological symptom of increased intracranial pressure.

6. The protein level in the cerebro-spinal fluid was high.

7. The ventricular estimation was of diagnostic importance.

The second case is of interest, as it shows a high degree of constant arterial hypertension in a man, aged nineteen years, with a supratentorial tumour and a calcified astrocytoma of the left occipital lobe, whose blood pressure returned to normal after the successful radical removal of the tumour. This case stresses the difficulty of diagnosis based on clinical symptoms, whereas the X-ray signs made a correct diagnosis easy.

Summary.

Constant arterial hypertension was present in two cases of brain tumour; one patient was aged forty-three years and had a meningioma of the posterior fossa; the other was aged nineteen years and had an occipital astrocytoma. After the successful removal of the tumours the blood pressure dropped to normal levels.

Reference.

(1) B. C. Meyer: "Neoplasm of the Posterior Fossa Simulating Cerebral Vascular Disease: Report of Five Cases with Reference to the Role of the Medulla in the Production of Arterial Hypertension", *Archives of Neurology and Psychiatry*, Volume XLV, March, 1941, page 468.

EYE DISEASES FOUND IN TASMANIA: AN EIGHT-YEAR SURVEY.

By J. BRUCE HAMILTON,
Major, Australian Army Medical Corps, Abroad.

I RECENTLY had the opportunity of reading the annual statistics of the Hobart General Hospital (now the Royal Hobart Hospital), published a little over eighty years ago. From that perusal two things stand out clearly: firstly, the great change in our nomenclature of diseases during the last eighty years, and, secondly, the vast change in disease ratio as compared with the statistics of 1940. It therefore occurred to me that, if only for historical purposes, a list of eye diseases found in Tasmania between 1931 and 1939 might be of interest, and with that object the following table (Table I) has been compiled. Such a list must have a personal flavour; but it gives a fair cross-section of ophthalmology in private practice in this island State, and it is interesting to compare this list of eye diseases with my tables of blindness, published in the *Transactions of the Ophthalmological Society of Australia*, Volume II, 1940, page 37. How can we understand blindness without understanding disease incidence?

Comments.

There are a few comments on this table which I should like to make—not new, but worthy of repetition.

Amblyopia ex Anopsia.—Are 236 cases of *amblyopia ex anopsia* necessary? I think a better public school and private school eye service would prevent this great visual loss.

TABLE I.

Eye Diseases Found in Analysis of 6,460 Cases in Tasmania, 1931 to 1939.

Number of cases of diseased conditions, 14,317. Number of individual diseases, 239.

Accommodation, paralysis of	34
Albinism	5
Amblyopia—	
Congenital	35
Ex anopsia	236
Functional	14
Anæsthesia of orbital nerves, supraorbital and infraorbital	3
Angioid streaks	4
Angioma, various types	12
Angioneurotic œdema	2
Anisocoria	90
Anisometropia	62
Anopia, quadrantic and hemi-	16
Anophthalmos	80
Aphakia	143
Argyll-Robertson pupil	5
Argyrosis	11
Atropine irritation	12
Birth injuries	29
Blepharitis—	
Simple	159
Ulcerative	7
Brow, abscess of	18
Canaliculus, laceration of	3
Cataract—	
Anterior polar	4
Blue dot	14
Complicated	49
Congenital	65
Diabetic	37
Glass-blower's	1
Lamellar	5
Senile	681
Traumatic	53
X-ray	1
Chalazion	113
Chorioid—	
Detachment of	1
Rupture of	5
Sarcoma of	11
Stretching of	46
Colobomata—	
(a) Of chorioid	4
(b) Of disk	3
(c) Of iris	1
(d) Of macula	1
Chorioiditis—	
Acute	9
Old	127
Tay's	64
Colour blindness	11
Conjunctiva, cyst of	10
Conjunctival abrasion	5
Conjunctival burn	10
Conjunctival foreign bodies	39
Conjunctival laceration	13
Conjunctivitis—	
Acute—	
(a) Bacillus unknown	90
(b) Staphylococcal and streptococcal	7
(c) Pneumococcal	9
(d) Koch-Weeks	9
(e) Allergic	7
(f) *Ophthalmia neonatorum*	13
Conjunctivitis—*Continued.*	
Angular	39
Chronic	243
Convergence—	
Excess	98
Insufficiency	184
Corneal abrasion	87
Recurrent	5
Corneal burn	12
Corneal foreign body	101
Corneal laceration	12
Corneal nebula	251
Corneal staphyloma	3
Corneal ulcer (see dendritic ulcer)	151
Cyclitis	14
Cyclophoria	11
Dacryocystitis—	
Acute	18
Chronic	79
Dendritic ulcer	13
Dermoid cyst of—	
(a) Brow	1
(b) Lid	1
(c) Limbus	1
(d) Orbit	1
Disk, optic, cyst of	2
Divergence insufficiency	115
Eales's disease	3
Ectropion	42
Electric ophthalmia	2
Embolism of central artery	3
Endophthalmitis	3
Enophthalmos	4
Entropion	7
Epicanthus	12
Episcleritis	10
Esophoria	206
Esotropia	172
Eserine irritation	9
Exophoria	418
Exophthalmos	26
Exotropia	92
Glaucoma—	
Acute	8
Chronic	78
Secondary	48
Glioma retinæ	2
Globe—	
Contusion of	38
Perforation of	62
Hæmorrhage—	
Retinal	38
Subconjunctival	61
Subhyaloid	3
Herpes ophthalmicus	17
Heterochromia iridis	9
Horner's syndrome	4
Hordeolum	180
Hyaloid, persistent	7
Hypermetropia	343
Hypermetropic astigmatism	1,949
Hyperphoria	107
Hyphæma	32
Hypopyon	11
Hypothyreoidism	3
Intracranial tumours	17
Intraocular foreign bodies	8
Iris—	
Atrophy of	5
Bombé	12
Iris—*Continued.*	
Sphincter rupture	7
Perforation of	5
Prolapse of	12
Iridocyclitis	55
Iridodialysis	8
Iritis—	
Acute	46
Diabetic	5
Old	27
Keratitis—	
Acne rosacea	11
Bullous	4
Disciform	3
Interstitial	20
E lagophthalmo	5
Marginal	55
Mustard gas	2
Neuro-paralytic	9
Profunda	11
Striate	8
Superficial	17
Superficial punctate	95
Vesicular	9
Keratoconus	32
Lachrymal fistula	3
Lashes, itinerant	8
Lens, dislocated	15
Leucoma	12
Lid—	
Abrasion of	4
Concretions	30
Contusion of	16
Coloboma of	5
Dermatitis of	19
Foreign body	6
Herpes of	5
Laceration of	9
Papilloma of	28
Retention cysts	19
Retraction of	18
Wart of	12
Macula—	
Massive hæmorrhage	2
Hole at	4
Megalocornea	10
Melanoma—	
Of iris	8
Of retina	25
Microcornea	5
Microphthalmia	5
Migraine	38
Miosis	4
Mixed astigmatism	654
Mydriasis	20
Myopia	95
Myopic astigmatism	1,108
Myotonic pupil	8
Myxœdema	3
Nævus, pigmented (various sites)	25
Nasal accessory sinus infection	23
No abnormality diagnosed	119
Nystagmus	60
Obstruction—	
Naso-lachrymal duct	56
Punctum and canaliculus	33
Occlusion, post-cerebral artery	11
Œdema of lid, insect bite	2
Opaque nerve fibres	14
Ophthalmia, sympathetic	2
Optic atrophy—	
Leber's	6
Primary	30
Secondary	13
Orbital cellulitis	2
Orbital foreign bodies	2
Over-action of inferior oblique	4
Panophthalmitis	3
Papillitis	24
Papillœdema	9
Paralysis—	
External rectus	25
Facial or orbicularis	25
Inferior oblique	1
Inferior rectus	1
Internal rectus	3
Superior rectus	13
Superior oblique	3
Third nerve	11
Third and fourth nerves	1
Third and sixth nerves	6
Phlycten	43
Phthisis bulbi	10
Pinguecula	14
Presbyopia	1,978
Pseudoneuritis	14
Pterygium	102
Ptosis	39
Pupillary membrane, persistent	12
Retinæ commotio	8
Retinal arteriosclerosis	176
Retinal degeneration, senile	105
Retinal detachment	61
Retinitis—	
Active	29
Albuminuric	9
Arteriosclerotic	35
Circinata	2
Diabetic	28
Old	42
Retinitis—	
Pigmentosa	13
Proliferans	13
Senile massive exudative	10
Retrobulbar neuritis	11
Scleritis	18
Siderosis bulbi	4
Socket, contracted	2
Stenosis, puncta and canaliculi	4
Symblepharon	6
Synchisis scintillans	13
Synechiæ, anterior	32
Thrombosis of retinal vein	31
Thyreotoxicosis	23
Trachoma	11
Trichiasis	45
Ulcer, rodent	7
Uveitis	9
Vitreous hæmorrhage	29
Vitreous opacities	161
Vitreous prolapse	2
Xanthalasma	4

Chorioiditis.—What is the ætiology of 127 cases of old chorioiditis, and what part in its production is played by the tubercle bacillus? I think it is probably greater than we suspect.

Exophthalmos.—The low incidence of exophthalmos in a country where goitre is endemic needs consideration when American reports of malignant exophthalmos come frequently to our notice. Are the American and Tasmanian diseases the same? I doubt it.

Hypopyon.—The low incidence of hypopyon, when we realize that this figure includes hypopyon from diabetic iritis, is noteworthy.

Retinal Vascular Disease.—The incidence of three cases of embolism of the central artery compared with 31 cases of thrombosis of the central vein calls for comment. The small figure for the former, I believe, is undoubtedly due to the low incidence of rheumatic endocarditis in Tasmania.

Pterygia and Rodent Ulcer.—One hundred and two cases of pterygia and seven of rodent ulcer of the lids give the island a good climatic record.

Birth Injuries.—The number of birth injuries (29) and 39 cases of ptosis are suggestive; they have been mentioned in my paper in Volume I of the *Transactions of the Ophthalmological Society of Australia.*

Congenital Defects.—That there are no cases of buphthalmos, one case each of aniridia and of coloboma of the iris, two cases of *glioma retinæ* and only four cases of coloboma of the chorioid, is noteworthy at least.

Cataracts.—The cataracts (especially the senile type) are a formidable list. When will a generous ophthalmologist leave sufficient endowment for research to establish beyond doubt the ætiological basis of this distressing senile disease?

Sympathetic Ophthalmia.—There are two cases of sympathetic ophthalmia, both of many years' duration.

Let us hope that soon this condition will be better understood, so that fewer eyes will be excised than at present. Until we do understand its ætiology, large scale excisions must continue to prevent its onset.

Venereal Disease.—Thirteen cases of *ophthalmia neonatorum* and 20 cases of interstitial keratitis are not excessive in an eight-year period; but it must be remembered that these figures are from private and not from hospital practice. It is the duty of the obstetrician to see that these diseases shall be stamped out altogether; it is his shame that they still exist.

Conclusion.

Perhaps, eighty years hence, some lone medical historian will find and peruse these figures. I hope he will then thank (as I do now) the National Health and Medical Research Council of Australia for its assistance in preparing these statistics. It is my hope at a later date to compare this civil list with one of eye diseases in the field of battle. They should make an interesting comparison.

Reports of Cases.

AN APPARENTLY HITHERTO UNRECORDED VARIATION OF THE HUMAN AORTIC ARCH.

By R. BRANSTER, W. F. HERLIHY, D. W. LAWSON and R. J. NOWLAND,
Medical Students.

(From the Department of Anatomy, University of Sydney.)

IN the Department of Anatomy of the University of Sydney, during 1941 a case of right-sided aortic arch with an unusual disposition of its branches was discovered by the writers of this article. We were unable to find mention of a similar variation in the literature examined.

The cadaver was that of a well-built man with well-developed muscles, who in later life had accumulated a considerable quantity of subcutaneous fat. According to the records of the department he had died from chronic myocarditis at the age of seventy-eight years.

The Aortic Arch.

The aorta arose from the base of the heart in the normal position. It ran upwards and slightly to the right, coming to be anterior to the bifurcation of the trachea. It arched backwards to the right of the trachea (Figure I), and then, passing behind this and the œsophagus, curved forwards on the left side before descending in the median plane towards the diaphragm. The pulmonary artery emerged anterior to the root of the aorta, and immediately bifurcated. The superior *vena cava* lay on the right of the ascending aorta. The left innominate vein crossed the aortic arch immediately inferior to and slightly overlapping the root of the right common carotid artery.

The Branches from the Arch.

The first vessel to arise from the aortic arch was the left common carotid artery; then came the right common carotid and subclavian arteries separately, and finally the left subclavian artery from the left forward curve of the arch (Figure II).

The left common carotid artery arose from the postero-lateral aspect of the ascending aorta on the left side, about one and a half inches above the left atrium. This artery ran to the left, backwards and upwards. It curved round the anterior and left lateral surfaces of the trachea, and continuing upwards and backwards, crossed anterior to the origin of the left subclavian artery. It continued towards the root of the neck anterior and very slightly medial to the subclavian artery and antero-lateral to the œsophagus.

The right common carotid artery arose from the anterior surface of the aortic arch about four inches from the heart. It ran upwards and a little laterally, on the right of the trachea, to the root of the neck. It was lateral to the trachea and right recurrent laryngeal nerve.

The right subclavian artery arose from the superior surface of the arch, about half an inch distal to the origin of the right common carotid artery. It ran upwards behind the right vagus and phrenic nerves, and disappeared behind the right *scalenus anterior*. It lay postero-medial to the right innominate vein and, continuing, crossed behind the right internal jugular vein from its medial to its lateral side.

The left subclavian artery arose from a large bulge on the left antero-lateral aspect of the descending aorta. It ran upwards and backwards, curving laterally behind the left *scalenus anterior* to enter the axilla over the first rib. It was posterior and slightly lateral to the left common carotid artery throughout it course in the superior mediastinum. It was lateral to the œsophagus and the left recurrent laryngeal nerve, lying in the groove between the œsophagus and the trachea.

Beyond the limits described above the courses of these arteries were normal.

The *ligamentum arteriosum* ran from the superior surface of the left pulmonary artery to the root of the left subclavian artery.

The Related Nerves.

The vagus nerve on the left descended from the neck, postero-lateral to the left common carotid artery, to lie between the common carotid and subclavian arteries as they emerged from the thorax. It crossed the left subclavian artery and then ran downwards and backwards on its lateral side to the posterior pulmonary plexus.

The left recurrent laryngeal nerve left the trunk of the vagus about three-quarters of an inch below the lower border of the subclavian artery. It hooked round behind the *ligamentum arteriosum* and ran upwards over the anterior surface of the root of the subclavian artery. Continuing upwards, it lay between the left subclavian artery and the trachea, and posterior to the left common carotid artery. Hence it ran upwards and slightly backwards until it reached the groove between the trachea and the œsophagus, and, keeping the same relation to the carotid and subclavian arteries and the trachea, reached the neck.

The left phrenic nerve, running downwards in the root of the neck, lay anterior to the left subclavian artery and posterior to the common trunk of the suprascapular and transverse cervical arteries, which arose from the thyreo-cervical trunk. In its upper thoracic course the nerve lay lateral to the subclavian artery; then it passed anterior to the lung root and ran downwards and backwards behind the pericardium to the diaphragm.

The right vagus lay half an inch postero-lateral to the common carotid artery in the neck. In the root of the neck it passed anterior to the right innominate vein. It continued behind the superior *vena cava* and lateral to the trachea, then it ran lateral to the root of the right subclavian artery and crossed the aortic arch to the right of the subclavian stem. It passed postero-medial to the arch of the *vena azygos* and so reached the right posterior pulmonary plexus.

The right recurrent laryngeal nerve emerged from the trunk of the right vagus, at the lower border of the aortic arch. It hooked around under the arch, half an inch superior to the arch of the *vena azygos*, and came into relation with the right side of the trachea. It passed upwards into the neck, in the groove between the trachea and the œsophagus.

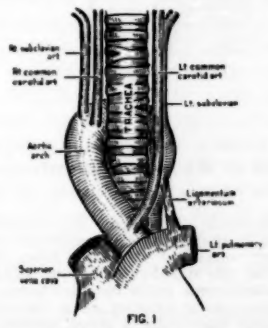


FIG. I

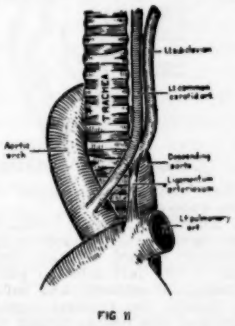


FIG II

The right phrenic nerve followed a downward course over the *scalenus anterior*. The nerve passed anterior to the subclavian artery and right costo-cervical trunk in the root of the neck, and then medial to the root of the internal mammary artery, which had a separate origin from the costo-cervical trunk. The nerve crossed the descending part of the subclavian artery from before backwards and finally passed lateral to the arch of the *vena azygos* and thence downwards to the diaphragm.

Normal Embryology.

The usual mode of development of the aortic arch system from which the vessels concerned are derived is illustrated in Figures III and IV.

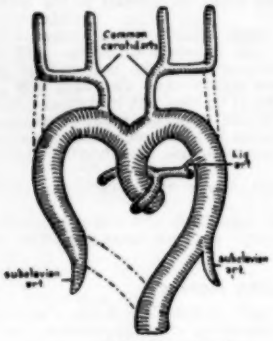


FIG III. (After Congdon)

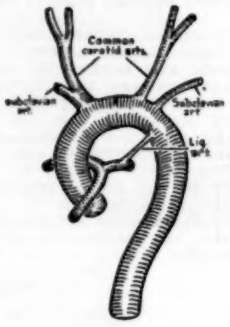
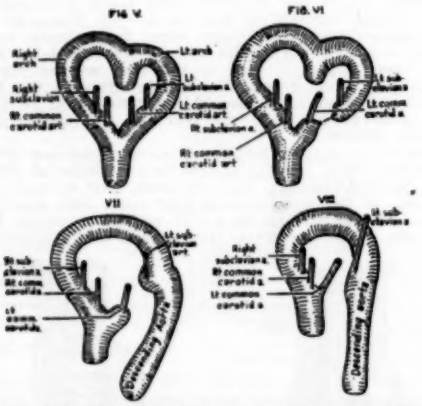


FIG IV.

A Probable Explanation.

Right-sided aortic arch has been described fairly frequently; here the difficulty is to explain the origin of the left subclavian artery. It would appear that for a time both fourth arches persisted, thus giving rise to a double aortic arch, as occurs in reptiles. From the proximal parts of each of these arches arose a common carotid and subclavian artery, as illustrated in Figure V, as in a case described by Arey. Next, the left fourth arch atrophied between the origins of the left common carotid and the left subclavian arteries. This stage is shown by Figure VI.

By a process of absorption the distal part of the left fourth arch was taken up into the dorsal aspect of the aorta until the left subclavian artery came to arise from the left lateral side of the descending aorta. In a similar way the proximal part of the left fourth arch was taken up into the ventral aspect of the aorta, and thus the left common carotid artery came to arise low down the ascending aorta. These final stages are illustrated by Figures VII and VIII.



Other Minor Variations in the Same Cadaver.

There were other small variations from normal in the same cadaver.

1. The left hepatic vein, instead of joining the inferior *vena cava* in the abdomen as it traversed its groove in the posterior aspect of the liver, emerged directly from the back of the left lobe to pierce the diaphragm by a special opening. It entered the inferior *vena cava* in the thorax.

2. Beside the normal hepatic artery arising as a terminal branch of the cœliac trunk there was quite a large accessory artery to the liver. It arose from the superior mesenteric artery, very close to its origin from the abdominal part of the aorta. This largely took the place of the right branch of the hepatic artery, which, although having a small right hepatic branch, was distributed mainly to the left lobe.

Summary.

1. A rare abnormality of the human aortic arch is described.

2. The normal embryology is indicated.

3. The probable explanation is suggested.

4. Two other anatomical abnormalities in the same subject are mentioned.

Acknowledgement.

Our thanks are due to Dr. A. A. Abbie, of the Department of Anatomy, University of Sydney, for his assistance in the preparation of this article and for the suggested explanation. Thanks are also due to Professor Burkitt for permission to report the case.

Reviews.

THE PRODUCTION OF ANTIBODIES.

THE first monograph of a series to be published by the Walter and Eliza Hall Institute of Research in Medicine and Pathology comes from the pen of F. M. Burnet,[1] who has long worked in the field of problems in immunity. He has gathered much evidence in relation to the production of antibodies and has put forth an interesting hypothesis which unifies some theories and links together many otherwise isolated observations.

Burnet accepts the premise that the specific effect of an immune serum is related to the globulin molecules which it contains, and further that it is the surface of those molecules which has been adapted to unite with an individual antigen. He then discusses the nature of the plasma proteins and the variations of the globulin fraction after the injection of chemical or of particulate antigens. In this section he includes many results of his own experiments in antibody titre after a single dose of antigen, and establishes the fact that antibody production continues at a decreasing rate after the antigen has disappeared from the circulation and that the rate of disappearance may be extremely slow, so that the production of antibody may be prolonged over a considerable period of time. There is also evidence that a second injection of antigen provokes a higher level of response than the first.

The author then considers the theories of protein structure and size and the intracellular molecules which must take part in the process of antibody formation in the cells of the reticulo-endothelial system. He discusses Bergmann's theory of molecular configuration and the symmetry of the polypeptide linkage. The suggestion is put forward that certain points in the molecule may have greater stability than others and may be the places at which additions to the molecule could be formed in "partial replica" of the parent molecule. He believes that this parent molecule acts as an enzyme and a pattern at the same time, and can build and liberate these partial replicas. Antigenic material then, taken into the cell, can modify the protein elaborated and cause the synthesis of the globulin molecule concerned in the corresponding immunity reaction. As the antigen is destroyed the cell protein which produced the modified antibody molecule may continue to produce the same pattern and liberate it into the blood or lymph.

Burnet draws attractive analogies between the enzyme activity of bacteria "trained" to ferment certain sugars and of tissue cells trained to produce antibody to a given nitrogenous antigen.

He concludes with a review of recent work in fields bearing on the subject and mentions problems yet to be solved.

The monograph is written from the point of view of the biologist, and of necessity more attention is devoted to biological than to chemical evidence; it may well be that advances in protein chemistry may sustain or modify this theory. In the present state of our knowledge, however, the author's theory coordinates the biological evidence on the activities of the reticulo-endothelial system and the chemical theories of molecular configuration in a useful way.

This is a monograph for a worker in the field of immunology and bacterial chemistry, not for the clinician or the inquiring amateur. The institute is to be congratulated on the steady constructive purpose behind the publication, which Dr. C. H. Kellaway expresses so clearly in his foreword. This monograph may well be called, in Wilfrid Trotter's happy phrase, "a due modicum of the vitamin of ideas" which is so necessary for the real growth of the body of science.

[1] "Monographs from the Walter and Eliza Hall Institute of Research in Pathology and Medicine, Melbourne; Number One: The Production of Antibodies: A Review and a Theoretical Discussion", by F. M. Burnet, M.D., Ph.D., with the collaboration of M. Freeman, B.Sc., A. V. Jackson, M.B., B.S., D. Lush, M.Sc.; 1941. Melbourne: Macmillan and Company Limited. Crown 4to, pp. 32, with diagrams.

The Medical Journal of Australia

SATURDAY, JANUARY 10, 1942.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: Initials of author, surname of author, full title of article, name of journal, volume, full date (month, day and year), number of the first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction are invited to seek the advice of the Editor.

THE SUPPLIES OF MEDICAL EQUIPMENT: CONSERVATION AN URGENT NECESSITY.

READERS of this journal are, or should be, aware of the activities of the Medical Equipment Control Committee. More than twelve months have passed since the publication of an address by Sir Alan Newton, Chairman of the Committee, on problems relating to the supply of medical equipment. Since that time a great deal has been done to meet the needs of the present emergency. Stocks have been husbanded, imports have been arranged and controlled, manufacture in Australia has been encouraged and steps have been taken in many instances to see that the most effective use was made of available stocks. At no time in 1941 did the need for economy become less; since the entry of Japan into the war it has become greater than ever. This will be quite obvious, and medical practitioners will be prepared to cooperate with those who are doing what they can to secure the maintenance of supplies. They will be able to do this the more easily if they are kept informed of shortages and advised from time to time of substitutes and of methods of making available material go as far as possible. The frequent dispatch of circulars is not really satisfactory; it is also wasteful of paper (an important consideration at present) and time-consuming. The Medical Equipment Control Committee will therefore forward periodically to this journal information for the guidance of practitioners; the first of such contributions is published in another place in this issue. It is hoped that those who read this journal will disseminate the information among those of their colleagues who do not read it, and among those who control hospitals and similar institutions visited by them. The Medical Equipment Control Committee wishes to defer, and if possible to avoid, the gazettal of regulations rationing the various items of medical equipment. This will be possible if the medical profession, knowing the relevant facts, will lend its full cooperation.

UROLOGY.

IN his autobiography recently reviewed in this journal Hugh Hampton Young refers to the pride that urologists may feel from the fact that their branch of medicine is the only one mentioned in the Hippocratic oath and he quotes the section:

> With purity and with holiness I will pass my life and practise my art. I will not cut a person for stone, but will leave this to be done by practitioners of this work.

Hippocrates made the patient his first consideration, for the oath continues:

> Into whatever houses I enter I will go into them for the benefit of the sick, and will abstain from every voluntary act of mischief and corruption . . .

Clearly in bygone ages there were those who made themselves expert in at least one urological procedure, and the Father of Medicine with the welfare of the patient uppermost in his mind will have no general meddling by the inefficient when skilled hands are forthcoming. With the enormous strides that have been made in surgery during the last half century, urology has perhaps made disproportionate advances; it has certainly emerged as a specialty whose members have signal opportunities of giving relief to human suffering. Sir Hugh Lett has recently pointed out[1] that in no branch of surgery has diagnosis been brought to such a high degree of accuracy as in the diseases of the urinary tract. In view of the claims of urology to an ancient foundation and in view of its latter day attainments, it is not inappropriate to take stock of our attitude to it and to those who take it as their sole life's work. Sir Hugh Lett's article, already mentioned, will be a help in this stocktaking, and the same may be said of a recent review by Hugh H. Young.[2]

Inquiry into the history of urology in Garrison's "History of Medicine" shows that from among the ranks of such "itinerants" as the Norsini who were skilful in hernia and lithotomy, and the Colots who cut for stone only, there was evolved the great Provençal surgeon Pierre Franco who was the first to perform suprapubic cystotomy in 1559. Garrison, however, quotes Lejeune as holding that the almost forgotten Francisco Diaz, body physician to Philip II, who in 1588 published the first treatise on diseases of the kidney, bladder and urethra, was the virtual founder of urology. But the whole of surgery, as it were, lay dormant until the coming of Joseph Lister and it is clear that but for his monumental work, modern urology, as we know it today, would not exist. At the same time, as Lett rightly insists, while we acknowledge our debt to Lister, we must not forget what we owe to physicists and biological chemists. Lister made operation safe; but physicists and chemists on the one hand gave us new methods of diagnosis and the means to assess kidney function so that we should know when to operate, and on the other made possible the elaboration of medical forms of treatment so that many urinary infections might be treated without operation. Though the sphere of urology is set within narrow limits, it calls to its aid many of the medical sciences, and Young is doubtless correct in his view of urology that there is no branch of medicine in which embryology, endocrinology, comparative

[1] Sir Hugh Lett: "Forty Years' Experience of Urology", *Proceedings of the Royal Society of Medicine*, August, 1941.

[2] Hugh H. Young: "Fifty Years' Progress in Urology", *The American Journal of Surgery*, January, 1941.

and regional anatomy, bacteriology and other forms of laboratory investigation play so important a role. To be a proficient urologist it is necessary for a surgeon not only to be skilled in the use of diagnostic methods and in the assessment of renal function, but also to have more than a bowing acquaintance with the medical sciences mentioned by Young. The scope of urology has grown with the methods used by its practitioners and the final boundaries of the subject have not yet been set. The discovery of X rays in 1895 was soon followed by their application to the problem of stone in the kidney, for in 1896 MacIntyre of Glasgow obtained the first positive radiograph of a renal calculus. That many years passed before radiography for stone in the urinary tract could be said to be firmly established is common knowledge. For long what is known as the plain skiagram had to suffice, but then came retrograde pyelography and finally in 1929 excretion urography. Of the last mentioned Lett states that it is difficult to over-estimate the important part it now plays in the diagnosis of surgical conditions of the kidney and ureter. He also points out that the modern urological surgeon would regard as unthinkable an operation on the kidney unless either retrograde pyelography or excretion urography or both had been undertaken. There is no need to enter on a discourse regarding the light that has been shed on obscure conditions of the renal tract by modern radiological investigation. It should perhaps be noted at this stage that cystography and urethrography, methods of investigation all too little used, must not be forgotten when radiography of the urinary tract is mentioned. Endoscopy of the urinary tract which began when Bozzini of Mainz and Frankfurt devised his *Lichtleiter* in 1807, has become a routine procedure with every urologist. The names of Nitze, Guyon and Albarran are amongst those prominently associated with the development of the cystoscope, and the accuracy of the observations made with it as well as the uses to which it can be put appear to the unskilled as almost uncanny. When we recall the introduction of the urea concentration test and of the indigo-carmine and the phenolsulphonephthalein tests we have no difficulty in appreciating their enormous value, especially when they are used in conjunction with endoscopic and radiological methods. The results of research into disease of the urinary tract and records of success in its treatment lead inevitably to the conclusion that urology is a special branch of medicine in the proper meaning of the term, that it is worthy of choice as a life's work and that more is needed in its devotees than mere proficiency in technique.

What of the recognition of urology as a specialty in Australia? It must be owned that the position is not entirely satisfactory. The late Robert Gordon Craig did splendid service to the specialty when he endowed the Department of Urology at the Royal Prince Alfred Hospital, Sydney. He not only established a department of urological service in a large teaching hospital, but he also provided for the training of Fellows in Urology, who on the completion of their training would adorn the specialty, and he put research in urinary tract disease on a solid footing. But in all States of the Commonwealth urological departments are not functioning in teaching hospitals as they should function. It is said of some general surgeons that they will not hand over special urological work to urologists and this for varied reasons. If the reason of their desire to combine urology and general surgery is the result of an honest opinion that their results are in every detail as good as those of the specialist in urology, they must be complimented for unusual ability and there is no more to be said. If they cannot with a clear conscience and in all self-honesty make this claim, they should do what lies in their power to see that the controlling bodies of hospitals should at least inquire into the attitude taken by the Father of Medicine. It would be much better to determine that no unskilled practitioner shall "cut a person for stone", but shall "leave this to be done by practitioners of this work" rather than that one sufferer should die or be maimed by the cutting.

Current Comment.

FLUORESCENCE IN THE DETECTION OF TUBERCLE BACILLI.

A QUICK and simple method for the detection of small numbers of tubercle bacilli in the sputum is an important requirement. The most painstaking search of a smear stained by the Ziehl-Neelsen method may fail to reveal tubercle bacilli although the organisms may be recovered from the same sample by culture methods or animal inoculation. Unfortunately these two procedures are very slow. Oscar W. Richards, Edmund K. Kline and Raymond E. Leach have recently described a method of demonstrating tubercle bacilli by the use of a fluorescent material in staining.[1] They point out that the spontaneous fluorescence of some substances was observed by Köhler in 1904, when he was experimenting in dark-field microscopy with ultra-violet light. The "fluorescence microscope" was introduced in 1911, but did not achieve any great distinction. Provazek discovered in 1914 that substances could be made to fluoresce by soaking in a fluorescent chemical. Kaiserling observed fluorescence of tubercle bacilli in 1917. Later he published observations on the fluorescent properties of other bacteria. In 1937 Hagemann noted the fluorescence of acid-fast organisms stained with berberine sulphate. In 1938 the same worker found that staining with auramin gave better results than staining with berberine sulphate. The value of this method was confirmed by various investigators, including Hermann, who found tubercle bacilli in a greater number of samples of sputum by the fluorescence technique than when the ordinary Ziehl-Neelsen method of staining was used.

The apparatus for microscopic examination of fluorescent material "consists essentially of light from a source rich in ultra-violet rays passed through a blue ultra-violet transmitting filter, reflected through the microscope from an aluminium-surfaced mirror and finally passed through a yellow contrast filter placed in the eye-piece". A "high amperage, low voltage, concentrated filament bulb is satisfactory" as the source of light. The filters absorb the rays that are not required, and the field appears dark. Organisms retaining the auramin stain appear as "self-luminous, golden yellow, bodies with a distinctive morphology". The organisms can be located with a sixteen-millimetre objective and a 20× eyepiece. They can be examined in detail with a four-millimetre objective and a 10× eyepiece.

A direct smear of the material to be examined is made on a glass slide and fixed by heat in the usual manner. A 0·3% solution of auramin in 3% carbolic acid solution is applied for two or three minutes. The slide is then washed with water. A solution of 0·5% hydrochloric acid and 0·5% sodium chloride in 70% alcohol is applied for

[1] *The American Review of Tuberculosis*, September, 1941.

one minute, then poured off; a fresh solution is then added for two minutes. This is to decolorize. The preparation is then rinsed and dried. Fresh sputum is preferable. If a preservative is necessary phenol gives the best results. Cresol reduces the number of organisms and causes fluorescence of itself. The concentration of auramin in the stain need not be accurate. In the preparation of the stain the auramin is dissolved by gentle heat. The solution tends to become cloudy on cooling; but this does not matter. After a number of weeks the stain loses some of its fluorescent properties. Smears of sputum known to contain tubercle bacilli should be stained and examined occasionally as a check on the stain's quality. If the light is strong enough, the findings by the four-millimetre objective may be checked by the use of an oil-immersion lens. Sandalwood oil should be used, as cedarwood oil is fluorescent. In this method the field appears grey. However, after a little practice the observer does not require to use the oil-immersion lens at all.

Richards, Kline and Leach have tabulated the results of some of their experiments. The figures they produce are striking. For example in 100 fields examined by the Ziehl-Neelsen technique 780 tubercle bacilli were found in one specimen as against 6,800 when fluorescence was employed. In other cases no organism was found by the Ziehl-Neelsen technique as against from five to 128 by fluorescence. It should be noted that the sizes of the fields are not the same, the ratio being 1·5 (Ziehl-Neelsen) to 7·0 (fluorescence). By restaining with the Ziehl-Neelsen method a number of preparations that had been stained by auramin, they found an occasional organism that had not been demonstrated by fluorescence. It is apparent, therefore, that all tubercle bacilli in a smear are not invariably revealed by either method. They conclude that fluorescence has the following advantages over older methods:

(*a*) The method stains more acid-fast bacteria than are stained by the Ziehl-Neelsen technique.

(*b*) Examinations can be made with high dry objectives covering fields which are larger than oil immersion fields as well as being simple and more convenient for use.

(*c*) A greater contrast obtains between the stained organism and other material present on the slide.

The last paragraph expresses what Richards, Kline and Leach regard as the most striking advantage of the newer technique. Acid-fast bacilli stand out and are readily recognized. This and the use of the ordinary high-power objective facilitate the investigation and make possible a great saving in time. The simplicity of the staining method also commends it.

The theory of microscopy by fluorescence is discussed by Emil Bogen.[1]

Ordinary microscopical visualization of bacteria depends upon the obstruction offered to the transmission of certain light rays by the stained or unstained organism. If the obstructing body is too small, the area of diminution or change cannot be distinguished from the remaining illuminated field, and so the organism is not seen. This definitely limits the possibilities of detection of minute forms by transmission microscopy.

Fluorescence microscopy depends upon the ability of certain substances to transform incident radiation into emergent waves of lower frequency. These longer waves in the region of the visible spectrum may be seen when other visible light is excluded by suitable filters. The points of light may be seen, however small, and visualization of minute forms with relatively low magnification is therefore possible.

Nonfluorescent structures, such as bacteria, may be made to fluoresce by impregnation with strongly fluorescent chemicals, such as certain dyes. Fluorescent staining results in the bacteria emitting light of a wave-length that will pass the eye filter, and so be seen in a field in which all other such light has been excluded.

Bogen recommends examination of the slides in a dark or shaded room. The fluorescent organisms may be seen with the low power of the microscope; but as they have to be distinguished from other objects—naturally fluorescent or retaining some stain—higher magnification is necessary for diagnosis. "The typical thin, bright, slightly curved rods" may be readily recognized at a magnification of 200.

This technique scarcely seems suitable for the medical man who occasionally uses a microscope. But it should be of great value to the bacteriologist. The search for tubercle bacilli by the Ziehl-Neelsen technique is often a wearisome business. The demonstration of the causal organism in tuberculosis is always desirable, however clear the clinical or radiological picture might seem to be; for without it diagnosis cannot be regarded as complete. In some cases the diagnosis must remain obscure until the presence or absence of the tubercle bacillus can be proven. Therefore any measure designed to facilitate the demonstration of the organism is to be commended. The technique described so well by Richards, Kline and Leach and by Bogen demands the careful investigation of every laboratory worker who is called upon the examine sputum and other material for the presence of *Mycobacterium tuberculosis*.

[1] *Ibidem.*

THE MENTAL HOSPITALS IN VICTORIA.

THE report of Dr. J. Catarinich, Director of Mental Hygiene for Victoria, covering the year ended December 31, 1940, has come to hand. The document, which is of some interest, loses much by the delay in its publication; a delay of almost twelve months is too much. The letter of the Director to the Chief Secretary, with which the report commences, bears the date August 18, 1941.

The first statement of the Director is important. During the twelve months under review the number of patients in residence in the Victorian mental hospitals increased by 194. This is attributed to two factors: a very low death rate and the increasing difficulty experienced by relatives retaining patients in their homes or providing private treatment owing to increasing taxation and higher costs of living. The cost of maintenance rose from £1 8s. 6d. per week per patient to £1 9s. 0¾d.; higher expenditure is expected before the end of the war. The total number of registered insane persons in the State on December 31, 1940, was 7,346. Fewer certified patients were admitted to hospital than usual, but the number of voluntary boarders was greater. As Dr. Catarinich states, the voluntary boarder method of obtaining treatment is most desirable, as in the great majority of cases it avoids the necessity for certification. The most notable forms of insanity among persons admitted to hospital were congenital mental deficiency (150 cases), *dementia præcox* (172 cases), delusional insanity (138 cases), senile and secondary dementia (184 cases). The names of no fewer than 1,046 persons suffering from *dementia præcox* remained on the books of the hospitals, "and this number is an indication of the serious problem presented by this mental disorder". The old complaint of overcrowding comes up again in this report. The increase in the number of resident patients has been largely responsible. The female divisions have been mainly affected. Some relief is expected as the result of an arrangement made with the authorities of the Ballarat Benevolent Home, who in their new building scheme have made provision for the accommodation of 100 patients from the mental hospitals. Dr. Catarinich recommends the erection of a new ward at Mont Park for the accommodation of aged and invalid types of female patient. The building programme undertaken by the Cabinet to replace obsolete buildings has not progressed as rapidly as Dr. Catarinich desires. He recognizes that we are living in abnormal times and that first consideration must be given to defence needs. He is cheering over one aspect—the "very undesirable conditions" existing in the male division of the Ballarat Mental Hospital will be completely removed at an early date. Final reference must be made to Dr. Catarinich's plea for the maintenance of a high standard of nursing among male attendants. He is in difficulties now owing to heavy enlistments, and he hopes that when the war is over nursing of the insane will not be regarded merely as an avenue for supplying positions to anyone who desires a job.

Abstracts from Medical Literature.

PATHOLOGY.

Monotreme and Marsupial Cerebral Vascular Patterns.

S. SUNDERLAND (*The Journal of Comparative Neurology*, August, 1941) has investigated the arrangement of the cerebral blood vessels in some marsupials and in the two monotremes. In regard to the marsupials he confirms the observations of American workers that the vessels form capillary loops. In both monotremes, however, the vessels terminate in a continuous capillary network similar to that found in the Eutheria.

Osteogenesis Imperfecta.

F. REGIS RIESENMAN AND WALLACE M. YATER (*Archives of Internal Medicine*, May, 1941) have made a thorough examination of seven families, 32 members of which were found to have *osteogenesis imperfecta*. In five of the families there is a definite hereditary tendency, while in the other two families with no such tendency the disease is represented in its isolated form. The ascendants, descendants and collaterals of the various families were studied, and 91 cases of the disease were found in a total of 255 members. Seven generations of one family, six generations of two families and five generations of another family were traced. The origin of the condition in one of the family groups was in Ireland, in 1790; in two family groups in Germany, one in 1735 and the other in 1770, and the remaining four in the United States of America, two in Virginia, dating back to 1808 and 1896, and the other two in the District of Columbia. The disease appears in three distinct forms: *osteogenesis imperfecta congenita*, *osteogenesis imperfecta tarda*, or the infantile type, and idiopathic osteopsathyrosis. The first two forms are congenital and include the presence of the blue discoloration of the sclera. *Osteogenesis imperfecta* is an hereditary and familial disease and is transmitted as a dominant Mendelian factor. It is an hereditary mesenchymal hypoplasia due to a disturbance of the gene or genes that determine normal mesenchymal development. The most important features representative of the clinical manifestations of the disease are blueness of the sclerotics (100%), brittleness of bones with multiple fractures (65%) and otosclerosis with progressive deafness (43%). The most common deformities are spinal curvatures, sabre-like deformity of the long bones and *pes planus*. Blueness of sclerotics may be the only manifestation of the disease, and when associated with a history of other manifestations in other members of the family group is pathognomonic of the syndrome. In the absence of obvious causes of blueness of the sclera they are pathognomonic even when such a family history is not obtainable. The intensity of the blueness of the sclera is not necessarily an index of the severity of the disease. Certain families have a predilection in regard to the frequency, severity and time of onset of otosclerosis. Certain families have a predilection in regard to the incidence of brittleness of bones with multiple fractures; but there is considerable variation in the occurrence of this factor even in individual families. With few exceptions there is a remission of the tendency toward fracture at the onset of puberty. Adults rarely show the Röntgenographic features of the disease. The osseous abnormality is the only reversible feature. Multiple fractures at the same site often result in deformity and incapacitation. The Röntgenographic appearance of the affected bones is characteristic, consisting of diminution in size of the diaphysis, great thinness of the cortex and presence of peculiar cross-striations. The early institution of proper orthopædic measures can do much to relieve the crippling nature of the disease. The economic background does not play a predisposing role in its appearance. Although there are three recognized forms of the disease, every case in this series is an example of the *osteogenesis imperfecta tarda*, or the infantile type, with the appearance of Lobstein's syndrome (brittleness of bones) as a factor in 57% of cases and Van der Hoeve's syndrome (deafness) as a factor in 43% of cases. The pre-natal and post-natal use of thymus extract has been suggested. Further progress in the treatment of the disease may depend on a better understanding of the principles of genetics and eugenics.

Non-Destructive Tuberculous Polyarthritis Verus: Tuberculous Rheumatism of Poncet.

IT is nearly forty years since Poncet described a type of tuberculous polyarthritis which resembles clinically an acute or chronic non-specific articular disease, especially rheumatic fever or rheumatoid (atrophic) arthritis. As the existence of such an entity is still controversial, David H. Kling and Max A. Levine (*Archives of Surgery*, May, 1941) present a detailed case history and a review of their own material and of the literature, and they come to the conclusion that a mild type of tuberculous polyarthritis does occur. It is due to a hæmatogenous infection of the joints from a visceral focus in a person who has a high tissue resistance. On the other hand, production of lesions in the joints by diffusible tuberculous toxins is denied. Only the demonstration of tubercle bacilli in the articular fluid or of tubercles in the synovial tissue is an irrefutable proof of the tuberculous causation of any articular lesion. It is not proved that the tubercle bacillus or its products are able to produce other than specific pathological changes. Confusion results from the assumption of many authors that only conditions in which non-specific lesions are present should be included under tuberculous rheumatism. The claim of Poncet and his followers that tuberculous rheumatism is a clinical entity without definite pathological background is not warranted. The lack of a clearcut clinical picture is just what makes the diagnosis uncertain. In the descriptions of Poncet and his followers tuberculous rheumatism mimics all types of acute and chronic arthritis. The tuberculous causation is in the majority of cases either unproved or doubtful. When pathological evidence has established the tuberculous nature of mild chronic polyarthritis, the condition should be designated by its chief characteristic, as non-destructive tuberculous polyarthritis. One can dispense entirely with the confusing term tuberculous rheumatism. In cases of polyarthritis which on clinical grounds is suspected of a tuberculous causation, a presumptive diagnosis of non-destructive tuberculous arthritis may be made.

Differential Sensitivity of Sarcoma and Normal Tissues to Temporary Arrest of the Circulation.

PETER SALZBURG AND HERMAN KABAT (*Archives of Surgery*, May, 1941) have investigated the effects of prolonged complete arrest of the circulation in an extremity of a rat bearing a chemically induced sarcoma. Although extensive necrosis of the tumour results when the circulation to the extremity is arrested up to the limit of subsequent survival of the animal, some viable tumour cells persist. The results of these experiments suggest that hopes of effecting a long-range cure of cancer based on the differential sensitivity of tumour cells and normal cells to the arrest of circulation may be ill-founded. On the other hand, further investigation of the susceptibility of other types of tumour to arrest of blood flow may conceivably lead to results of practical therapeutic value.

The Follicular Type of Malignant Lymphoma.

EDWARD A. GALL, HARVEY R. MORRISON AND A. THORNTON SCOTT (*Annals of Internal Medicine*, May, 1941) report on a series of 63 cases of the follicular type of malignant lymphoma that have come under their observation during a period of more than twenty years. In their own material follicular lymphoma has been manifested by moderate to enormous enlargement of lymph nodes with replacement of the normal architecture by multiple follicle-like nodules of varied size and approximation. Normal sinus structure was obscured and the capsule frequently encroached upon. Invasion of perinodal tissues was not common, but when present exhibited follicle-like arrangement even beyond the confines of the lymph node. Review of the sections revealed two fundamental structural variations. One consisted of closely packed, rounded or oval-shaped, follicle-like nodules of inconstant size but similar intrinsic morphology. The intervening tissue consisted only of thin strands of small lymphocytes or compressed reticular substance. The other type also consisted of innumerable widely distributed similar follicles. Instead of lying in immediate proximity to each other, however, they were separated by broad homogeneous masses of small lymphocytes. The follicles seemed set, more or less, in a lymphocytic matrix which was otherwise characteristic of the so-called lymphocytic form of lymphosarcoma. In these the architecture of the node appeared to be more completely obliterated by the combined processes. A distinct revision of fibrillar elements appeared. The reticulum meshwork surrounding each nodule was distorted and condensed by follicular expansion. The normal loose network with broad polygonal pulp spaces became compressed and the interreticular crevices were elongated and narrowed. The disappearance of the sinuses appeared to be the result of the displacement of reticular substance and pulp content. Despite the condensation of fibrils, fusion and collagen formation did not occur. All other types of lymphoma produced destruc-

tion of reticulum with the reappearance in some of scanty disconnected fibrils and in others of a fibrous stroma with no resemblance at all to the normal architecture. Ordinary hyperplasia caused no significant reticular rearrangement. This feature was, therefore, of considerable morphological diagnostic value. The authors have further divided the condition into four sub-groups according to the degree of differentiation observed. In only one of their cases did they find transition of the follicular process into what appeared to be another form of lymphoma. Clinical manifestations have been compared with those observed in 507 cases of known malignant lymphoma of varied types exclusive of follicular disease. Certain characteristic features have become evident. Individuals with the follicular process have exhibited initial symptoms at a much later period of life. The prognosis with regard to duration of the disease has been considerably longer with this group than with the remainder of the lymphomata. Constitutional manifestations and visceral involvement have been less frequent, and abnormal hæmatopoietic phenomena unusual. The apparently greater frequency with which retroperitoneal nodes become involved was impressive, and the proportion of those cases with chylous ascites was considered to be of some diagnostic import. All of these factors have caused the authors to conclude that follicular lymphoma, although a form of malignant disease of lymphoid tissue, has a distinct identity, with many characteristic clinical and structural features.

MORPHOLOGY.

Relation of Müllerian and Wolffian Ducts.

P. GRUENWALD (*The Anatomical Record*, September, 1941) has attempted to arbitrate between the two differing views as to the origin of the Müllerian duct by studying the development of the two ducts in chick and human embryos. He was unable to determine definitely whether the Müllerian duct is formed by splitting off from the Wolffian, in accordance with the older view, or whether the Müllerian duct develops independently of the Wolffian, as stated in the more recent work. He points out, however, that there is a very close correlation in the development of the two ducts, and feels that the older view demands serious reconsideration. He illustrates his contention by reference to a number of developmental anomalies.

Carotid Sinus Nerve Surgery.

D. SHEEHAN, J. H. MULHOLLAND AND B. SHAFIROFF (*The Anatomical Record*, August, 1941) have restudied the innervation of the carotid sinus with a view to determining whether simple division of the carotid sinus nerve (of Hering) is a practicable proposition in place of the extensive periarterial neurectomy usually advocated. The authors confirm previous observations that the sinus receives fibres always from the carotid sinus nerve, generally from the vagus, frequently from the sympathetic and rarely from the hypoglossal. The carotid sinus nerve arises from the glosso-pharyngeal close to the base of the skull, rarely more than four centimetres away, and runs down on the internal carotid artery, usually on the anterior aspect, but sometimes on the antero-medial or antero-lateral side, to the carotid sinus, whence one or more fibres can generally be traced to the carotid body and intercarotid plexus. The vagal contribution comes from the nodose ganglion or the pharyngeal branch and usually passes lateral to the internal carotid artery to join the carotid sinus nerve high up; in no case was the communication within three centimetres of the carotid bifurcation. Communication with the sympathetic is effected through a loop from the superior cervical ganglion and usually runs medial to the internal carotid artery, chiefly via the intercarotid plexus. An intercarotid plexus was found in every case; it receives vagal fibres, generally from the superior laryngeal nerve, and sympathetic fibres from the superior cervical ganglion. In view of the ease with which the carotid sinus nerve can be isolated, the authors conclude that division of the nerve is a feasible and preferable alternative to extensive periarterial stripping in the treatment of carotid sinus syncope.

Number and Volume of Human Blood Cells.

A. L. GRAFFLIN (*The Anatomical Record*, September, 1941) has calculated the total number of each kind of cell in the blood, and the volume each kind would occupy. The figures are only approximate. There are some 25,000,000,000,000 erythrocytes which occupy a volume of 2,000 cubic centimetres. Blood platelets number some 1,500,000,000,000, with a volume of 6·29 cubic centimetres. Neutrophile cells total 21,000,000,000, with a volume of 11·00 cubic centimetres; eosinophile cells are 750,000,000, with a volume of 0·29 cubic centimetre; basophile cells 150,000,000, with a volume of 0·06 cubic centimetre; lymphocytes are 6,300,000,000, with a volume of 1·13 cubic centimetres; and monocytes number 1,800,000,000, with a volume of 0·94 cubic centimetre.

Localized Thickening of Venous Walls.

C. TEDESCHI (*The Anatomical Record*, February, 1941) records a hitherto unrecorded localized thickening in the wall of a vein adjoining an artery. The observations were made upon humans, mostly between forty and seventy years of age, and some younger—down to newly born. The arterial side of the vein may be more than twice as thick as the opposite side, and the thickening is due chiefly to an increase in the adventitial muscle. Such thickening is uncommon under the age of thirty years, but increases progressively thereafter. On the other hand, the thickening may gradually disappear when the interposition of fatty or fibrous tissue reduces the intimacy of arterio-venous contact. Such veins as the subclavian and jugular, which run more or less independently of the corresponding artery, do not betray the thickening.

Dentinal Fibres.

G. BEVELAND (*The Anatomical Record*, September, 1941) has studied the development of the dentinal fibre system in a number of mammals, including man. The first discernible fibre formation is a fibrous reticular membrane which is continuous with the fibres of the dental sac. The membrane occurs in the periphery of the pulp, orientated parallel to the contour lines of the tooth. As the odontoblasts appear, the fibres of the membrane become radially arranged; additional fibrils from the pulp penetrate the odontoblast layer, splay out and extend to the dentino-enamel junction. With increase in dentine the fibres become rearranged: first, there is a condensation of radial fibres within the intertubular areas, and secondly, some fibres become disposed transversely or tangentially in relation to the dentinal tubules. The fibres in a mature tooth, although more numerous, do not differ materially from those in the newly erupted tooth. Primary dentine is laid down in reticular fibres throughout its development; there are no differences of origin between the fibres of the mantel and circumpulpal zones, but those in the mantel zone are thicker.

"Milky Spots" in the Pleura.

R. L. MIXTER (*The American Journal of Anatomy*, September, 1941) has studied the incidence and structure of "milky spots" (*taches laiteuses*) in the pleuræ of several mammals, including man. He found the spots constantly in every animal examined beyond a certain period of growth. The spots are variable in number and distribution, but occur most frequently in the retro-cardiac mediastinal pleura, also in the dorsal and ventral mediastinal and the pericardial pleura. The spots in the mediastinal pleura increase in number and size from birth to maturity. Some of the spots occurred in avascular areas, but the great majority were in intimate association with blood-vascular networks. The spots in human newborn are supplied only through arteries and veins; they have no lymphatic drainage. The venules draining, or close to, the spots—even as small as 0·1 millimetre in diameter—contain typical valves. Structurally the spots comprise mainly accumulations of macrophages; the overlying mesothelium is usually unchanged, but may show some stratified thickening during development and growth. Because of their structure the author prefers the name "macrophagal foci" for the spots.

Movements of Subtalar and Transverse Tarsal Joints.

J. T. MANTER (*The Anatomical Record*, August, 1941) presents an analysis of the movements possible between various tarsal bones of the human foot. The observations were made upon the feet from cadavers by means of a specially designed apparatus. The author considers the observations reliable, since the character of joint movement depends upon the shape of the articular surfaces. He found a screw-like movement at the subtalar joint—right-handed on the right side, left-handed on the left. The transverse tarsal joint permits the fore part of the foot to move in a longitudinal screw-like manner, the reverse of that at the corresponding subtalar joint. The transverse tarsal joint may also move about a second axis, which lies obliquely, to produce combined dorsiflexion and abduction of the fore part of the foot, causing a marked change in the longitudinal arch. Appropriate rotation in all three axes of the principal tarsal joints produces the position characteristic of flat-foot.

British Medical Association News.

SCIENTIFIC.

A MEETING of the Victorian Branch of the British Medical Association was held on September 17, 1941, at the Women's Hospital, DR. W. G. CUSCADEN, chairman of the honorary medical staff of the hospital, in the chair. The meeting took the form of a number of clinical demonstrations.

Recovery of Kidney Function after Transplantation of the Ureter.

DR. HAROLD MOORE showed some X-ray films taken after the intravenous injection of "Uroselectan" in a case of injury to the left ureter. The injury was sustained during a difficult operation for the removal of the uterus. It was not known that any injury to the ureter had occurred until about four days after the operation, when there was a leakage of urine from the vagina. The films were shown to indicate the possibility of conserving the kidney in these cases. The first series of films, taken three weeks after the operation, showed no evidence of excretion of dye on the left side. The next series, taken eight weeks later, again showed no evidence of excretion on the left side, although leakage of urine into the vagina was still occurring. At this stage an operation was undertaken, at which the lower end of the ureter was freed and transplanted into the bladder. The final series of films, taken about four and a half months after the plastic operation, revealed a slightly dilated renal pelvis and an apparently normal ureter. The excretion of dye at this stage was equal in rapidity and density on the two sides.

Dr. Moore said that the films were shown to illustrate the recovery of function in a kidney after the removal of a partial obstruction to the ureter. In this case relief was obtained by transplantation of the ureter into the bladder. The question of nephrostomy was discussed. Nephrostomy had not been carried out in this case, as it was thought that the kidney function was not seriously interfered with or there would not have been such free leakage of urine into the vagina. The necessity for allowing an adequate period to elapse before attempting repair was also stressed. This was partly due to the fact that in a small number of cases spontaneous cure would result, but more particularly due to the need for avoiding operation on œdematous and friable tissues.

Myomectomy.

DR. JOHN GREEN demonstrated from lantern slides certain aspects of myomectomy under eight headings, as follows.

1. A rubber tourniquet applied round the "neck" of the uterus controlled hæmorrhage. This prevented blood loss and allowed precise suturing.

2. Multiple uterine incisions should be avoided. The number of incisions should be small, in order to reduce the risk of post-operative obstruction from adhesions. As many fibroids as possible should be extracted through one uterine incision. The suture lines should be peritonealized by bringing over the round ligaments, by "advancing" the utero-vesical fold towards the fundus and by covering them with omentum.

3. With regard to infection, Dr. Green said that there was a greater tendency to morbidity with myomectomy than with hysterectomy. This might arise from tissue necrosis due to over-suturing and perhaps to the fact that an opening was made into the uterine cavity, although some surgeons opened into the cavity to facilitate drainage from the myoma bed. Predisposing causes of infective complications were sloughing, associated salpingitis and previous trouble from a septic abortion, a confinement *et cetera*. Infection was also likely if the patient's hæmoglobin value was low and if plugging had been necessary to control hæmorrhage. All these factors had to be considered, especially when the balance between hysterectomy and myomectomy was being weighed.

4. With regard to fibroid polypus at the os, Dr. Green said that it might be quite simple of management; but there were two traps, the sessile polypus and inversion.

5. The pedunculated polypus could be dealt with by twisting, snaring or cutting; but it was wise to sound the uterus carefully as a preliminary, to be sure that the growth was not a sessile submucous myoma presenting at the os. Such a case required adequate preparation for a possible vaginal hysterotomy.

6. A casual diagnosis might overlook a chronic inversion of the fundus, with disastrous results if the apparent attachment was cut off. Less obvious was the "traction inversion" brought about by pulling on an actual polypus; Dr. Green remarked that it would be embarrassing to find two "stalks" to one polypus after its removal.

7. Dr. Green went on to say that combined abdominal and vaginal fibroids were best managed in two stages. Particularly if the fibroid presenting to the vagina was sloughing there was a real risk of peritoneal infection by a one-stage procedure. The polypus should be removed (and this could often be done with narcosis induced by morphine and hyoscine) and the abdominal operation should be performed after a few days. This procedure also applied to hysterectomy in similar circumstances.

8. In pregnancy the risk of really serious trouble from degeneration was not great, and therefore it was better to evade myomectomy. When labour was obstructed by fibroids, the ideal operation would be myomectomy combined with Cæsarean section. Unfortunately there was more risk with Cæsarean myomectomy than with simple Cæsarean section or Cæsarean hysterectomy, particularly as the fibroids which caused obstruction were the less accessible ones. It was probably better in the first instance to be content with Cæsarean section alone. When the patient had accumulated her family the story could be completed with a gynæcological operation of myomectomy and sterilization.

The Effect of Intrauterine Malnutrition on Extrauterine Life.

DR. KATE CAMPBELL discussed the effects of fœtal malnutrition on the post-natal infant; she said that they were becoming increasingly obvious.

Dr. Campbell said that fœtal malnutrition could be produced in different ways, and she went on to discuss the most obvious. The first was a deficient maternal diet during the pregnancy, and Dr. Campbell described a clinical history to illustrate its effects. The patient was pregnant for the fouth time. During her pregnancy the diet consisted of white bread and butter, pies, crumpets, saveloys, meat, potatoes and tea, and she gained three stone in weight. She suffered from severe cramps in the limbs, with burning and numbness in the hands and "pins and needles" in the fingers; she developed weakness in the wrists and hands, so that she was unable to cut the bread or wring out the clothes. She went into labour at full term and had an ante-partum hæmorrhage for which no obvious obstetrical cause could be found. She also lost thirty ounces of blood *post partum*. The infant on the first day of life suffered from melena. It was treated with injections of "Kapilin" (the vitamin *K* analogue), and as the melena continued the baby was given an intramuscular injection of whole blood; it was not severely ill. The mother, when seen two or three days after labour, had patchy impairment of sensation in the hands and forearms and still complained of the symptoms which had been present during pregnancy. She was given injections of thiamin on alternate days and a full diet, and by the eighth day she had lost her symptoms.

Dr. Campbell remarked that in the case under discussion the mother showed evidence of peripheral neuritis due to lack of vitamin *B*. The ante-partum and post-partum hæmorrhages appeared to be due to a low prothrombin level in the blood brought about by insufficient vitamin *K*. It had been proved that hæmorrhagic disease of the newborn, of which *melena neonatorum* was the commonest manifestation, was due to the infant's prothrombin level (always below normal in the first three days of life) falling below the safe limit, so that spontaneous hæmorrhage occurred. In the case under discussion the mother showed evidence of lack of vitamin *B* and vitamin *K* and the infant showed evidence of lack of vitamin *K*.

Dr. Campbell went on to discuss anorexia in pregnancy. She said that this was often present in association with nausea. Many mothers who suffered severely from heartburn also voluntarily limited their diet most drastically in an attempt to avoid this symptom. Even though the diet was adequate, the actual food intake in both instances was not infrequently insufficient. If vomiting was pronounced, severe deprivation to the mother and consequently to the fœtus must ensue. In some instances even the intake of a satisfactory diet might be associated, in the absence of obvious disease, with poor nutrition. It was possible that in such cases there was some disturbance of the absorptive mechanism, and the fœtus of such a mother would be imperfectly nourished.

Dr. Campbell further said that post-maturity operated powerfully, introducing interference with the nutrition of the fœtus. It was well known that in the last two months of pregnancy the placenta began to undergo degenerative changes. If the fœtus was retained in the uterus after full term it was nourished by a degenerating and dying placenta and the fœtus must suffer. If it was retained too

long, the fœtus would die *in utero*; if it was born before this calamity, the baby might show obvious evidence of its perilous state. Such infants, in addition to showing the well-known post-mature condition of the skin, might be feeble, drowsy, lethargic and "bad feeders", easily exhausted before obtaining sufficient food; they had a feeble cry, rather like that of a premature baby. They frequently required devoted and skilled nursing to ensure their survival.

In those cases in which the placenta was small and undeveloped, the child was usually puny. It might be stillborn, or if born alive it was frequently frail and difficult to rear. In *placenta prævia* the mortality rate was notoriously high. Whilst some of the deaths had to be attributed to the effects of hæmorrhage, others were probably due to the poor placental development that occurred when the placenta was attached to the lower uterine segment.

Dr. Campbell said that in a study of the effects of intra-uterine malnutrition on the infant, it was at present possible only to appreciate those effects when the defects gave rise to some symptom or sign that was clinically obvious. It was not possible to assess those when the biochemical changes did not express themselves in signs visible to the clinical oberver; yet such conditions must be present. All clinicians were familiar with the infant who died in the neonatal period for no obvious cause and for whose death the pathologist was also unable to find a satisfactory cause of death at the post-mortem examination. There was no obvious anatomical cause, and the death must be due to metabolic disturbances. Further research in this field was urgently required. By the elimination of deaths from these causes the high mortality rate in the first week of life could be materially reduced. It was also possible that many of the obscure degenerative conditions which arose later in life might have their origin in faulty nutrition when the tissues of the fœtus were developing; the result would be an abiotrophy of organs, or symptoms.

Bacteriological Investigation in Clostridium Welchii Infection.

DR. A. M. HILL and MISS H. M. BUTLER discussed the growing importance of bacteriological investigation in the management of *Clostridium welchii* infections. Dr. Hill and Miss Butler pointed out that the two chief clinical types of *Clostridium welchii* infection following abortion and childbirth were those associated with jaundice and those associated with collapse. Cases associated with jaundice were most often the result of criminal abortion. The patient developed jaundice which rapidly deepened, and in the gravest cases her colour became bronze or mahogany; hæmoglobinæmia and hæmoglobinuria were also present. The pulse rate steadily quickened and the condition soon passed into one of peripheral circulatory failure, with subnormal temperature, falling blood pressure, running pulse, cold, clammy, cyanosed extremities and terminal dyspnœa. The patient died, usually retaining a clear consciousness to the last. Cases associated with collapse were a more difficult clinical problem, as varying degrees of collapse might result from a number of causes. When collapse was due to *Clostridium welchii* infection the patient had a steadily rising pulse rate, pallor and a general feeling of well-being. Insidious collapse occurred and soon passed into peripheral circulatory failure and death.

It was emphasized that if there was to be the slightest chance of successful treatment in these desperate cases, diagnosis must be early. It was at that point that bacteriological investigation had become of increasing value. In the experience of Miss Butler and Dr. Hill, examination of a smear from the cervix had proved the swiftest and most reliable method for the early detection of severe *Clostridium welchii* infections. In grave and fatal cases examination of the smear revealed heavily capsulated bacilli and damage to the leucocytes.

Dr. Hill and Miss Butler then showed three groups of clinical histories. All the patients referred to had been admitted to hospital during the previous six months.

The first group consisted of one case associated with jaundice. The patient, who suffered from a septic incomplete abortion after "taking pills", had obvious signs of pelvic inflammation. On the fifth day after her admission to hospital she developed jaundice, which steadily deepened for three days and subsided after another four days. At its onset the combination of jaundice with a rapid pulse rate made it essential to consider *Clostridium welchii* infection, and specific treatment was provisionally ordered. But immediate examination of the cervical smear failed to reveal *Clostridium welchii*. A blood film a few hours later revealed basophilic stippling. The patient was practically edentulous, and no blue line was seen on her gums. The final diagnosis was lead poisoning and pelvic cellulitis.

The second group consisted of four cases associated with collapse. The first case was one of toxæmia of pregnancy, followed by tubal induction of labour and intra-partum eclampsia. Thirteen hours after delivery this patient suffered from pallor, tachycardia and collapse, and *Clostridium welchii* infection was suspected. But examination of the cervical smear failed to reveal *Clostridium welchii*. The pulse rate fell after twenty-four hours and recovery was uneventful. The remaining three cases were examples of criminal abortion, in each of which interference had occurred a few hours before the patient's admission to hospital. Each patient showed pallor, shock and signs of collapse at or shortly after her admission. In the first of the three cases *Clostridium welchii* was not present in the cervical smear and recovery was uneventful. In the second case *Clostridium welchii* was present in the cervical smear; but the smear did not resemble that seen in grave infections and recovery was uneventful. In the third case *Clostridium welchii* was present in the cervical smear, which was of the type seen in grave and fatal infections. Specific treatment was not ordered and the patient died twenty hours after her admission to hospital.

The last group consisted of four cases of septic incomplete abortion, in which *Clostridium welchii* was recovered from the blood. In each case at some stage high fever and a rapid pulse rate were present; the total duration of fever lay between two and four days. In each case *Clostridium welchii* was present in the cervical smear, which, however, was not of the type seen in grave infections, and the organisms recovered from the blood differed morphologically and culturally from those causing grave infections. Not one of these patients presented the clinical features characteristic of *Clostridium welchii* infections, and all made an uneventful recovery.

It was pointed out that these clinical histories were presented to emphasize the fact that the mere recovery of *Clostridium welchii* from the body tissues and fluids did not of itself indicate severe *Clostridium welchii* infection. It was essential to stress this fact, for occasionally reports were received from overseas of similar cases in which *Clostridium welchii* had been recovered from the blood and in which recovery had occurred after treatment directed to that infection; credit for recovery had then been attributed to the treatment. Dr. Hill and Miss Butler pointed out that cases such as those last described created no problem of treatment, and recovery bore no relationship to any particular form of therapy.

The Management of Occipito-Posterior Presentations.

PROFESSOR R. MARSHALL ALLAN discussed the ætiology and diagnosis of occipito-posterior presentations and referred in detail to various signs which were of value in the estimation of the probable outcome of the labour. Treatment in general was discussed and illustrated by the records of 1,118 cases at the Women's Hospital. Spontaneous rotation occurred in 72% and manual rotation was required in 4%; 24% of patients were delivered with the fœtus persistently presenting in the occipito-posterior position. The incidence of forceps delivery in the series was 9%, as compared with 6·7% for all deliveries. Three maternal deaths occurred; but in none of the deaths could the fatal result be attributed to the presentation. There was a fœtal mortality rate of 2·5% in cases in which spontaneous rotation occurred, as compared with one of 10·6% when failure to rotate had been noted. The morbidity rate of both groups was 3·4% and 4·2% respectively.

Professor Allan advocated the use of sedatives during labour and the desirability of leaving the patient alone as long as her condition and that of the fœtus permitted, or until examination revealed that the progress of labour was at a standstill. When in doubt about any delay in labour, especially when it was unexpected, he did not hesitate to examine the patient under anæsthesia.

Placenta Prævia.

DR. W. D. SALTAU made a survey of cases of *placenta prævia* treated at the Women's Hospital during the preceding two years. There had been 52 cases; none of the mothers died and 24 infants were stillborn or died. The situation of the placenta was central in 15 cases, lateral in 20 and marginal in 17. Treatment was by Cæsarean section in 20 cases, by the application of Willett's clamp in four cases, by bipolar version in seven cases and by miscellaneous measures, such as rupture of the membranes, the application of an abdominal binder and the administration of pituitrin *et cetera*, in 21 cases.

In regard to the type of *placenta prævia*, Dr. Saltau stressed the difficulty of diagnosis in some cases. When the cervix admitted only one finger it was possible that the

edge of the placenta might be situated just inside, and if it were possible to insert two or three fingers the type might be really marginal. However, all cases in which the placenta was covering the internal os were classified as "central". Included in the twenty cases in which Cæsarean section was employed were 13 of the central type and seven of the marginal type. With regard to the indication for Cæsarean section in *placenta prævia*, Dr. Saltau considered that in all cases of central *placenta prævia* and in certain marginal types associated with much bleeding, and when the baby was anywhere near maturity, Cæsarean section should be performed. In the central type, at about thirty to thirty-two weeks, it was sometimes a little difficult to make a decision, as the safety of the mother was the primary consideration. If the mother was exsanguinated, Dr. Saltau thought Cæsarean section was indicated; but when the mother was in better condition more conservative treatment, such as bipolar version, might be employed. One had to remember that version in the case of the small baby was sometimes not easy, and the extra manipulation in the presence of exsanguination might be prejudicial.

Referring to the fœtal mortality rate after Cæsarean section, Dr. Saltau said that ten of the babies survived, three were stillborn and seven died during the puerperium. Prematurity was certainly the chief factor in the fœtal mortality rate associated with *placenta prævia*; but even some of the larger babies died, so that the exsanguinated condition of the mother also played some part. The application of Willett's clamp appeared to have rather gone out of favour, as this method was employed in only four cases in the series quoted. All four were of the marginal type, and three of the babies were stillborn. One of the advantages claimed for the use of Willett's clamp was that it ensured more living babies; but this belief had not been borne out in the series under discussion. The clamp was sometimes difficult to apply and occasionally was pulled off. Bipolar version was also much less common than formerly. Its chief disadvantage was the high fœtal mortality rate, as evidenced by the fact that in all seven cases in which it was performed the babies were stillborn.

An interesting feature was that in only one case of the series was the vagina packed. Dr. Saltau considered that on account of the great risk of sepsis, packing was permissible only as an emergency measure—for example, to enable the patient to be transported to hospital, or possibly in the uncommon case of the closed cervix as a preliminary to further treatment later. Included in the miscellaneous treatments were such measures as artificial rupture of the membranes and the application of an abdominal binder, possibly supplemented by small doses of pituitrin. Dr. Saltau stressed the inadvisability of rupturing the membranes as a method of treatment in the absence of labour pains, unless as a preliminary to other treatment. This measure was most useful in the more favourable lateral type of *placenta prævia*, when the patient was in labour and the cervix was dilating. Then the head was allowed to settle down onto the bleeding site, and this effect could be improved by a tight abdominal binder and the administration of small doses of pituitrin.

Dr. Saltau thought a pleasing feature of the series was the low maternal morbidity rate. Of the total of 52 patients, only 12 were "morbid" according to the British Medical Association standard. Of the 12, nine had had Cæsarean section; but most of the illnesses were mild and lasted from two to five days.

In conclusion Dr. Saltau emphasized the value of blood transfusion when the bleeding was severe. In all cases of ante-partum hæmorrhage in which severe bleeding was considered probable, the mother's blood was typed on her admission to hospital and a donor was obtained, or if one was not available resort was had to the "blood bank". Fourteen patients in the series received a blood transfusion, and this undoubtedly was an important factor in avoiding any maternal mortality and in the low morbidity rate.

The Lateral View in Obstetrical Radiography.

DR. COLIN MACDONALD discussed the value of the lateral view in obstetrical radiography; his talk was illustrated by lantern slides. He said that the standard postero-anterior view of the pregnant woman had been for many years in constant use at the Women's Hospital as an adjuvant to obstetrical diagnosis; often, however, it had to be supplemented by one or more lateral views. A lantern slide of a postero-anterior view was shown, in which only with difficulty could a fœtus be discerned; the difficulty was due to the fact that the small fœtal skeleton overlay the dense and obscuring shadows of the lumbar vertebræ. The most careful scrutiny had failed to elicit the important feature of this fœtus; however, in the lateral view anencephaly was shown for all to see. This "standard" lateral view was made with the central ray passing through the middle of the abdomen on a level with the upper limits of the iliac crest. Dr. Macdonald went on to say that it was not to the "standard" lateral view that he wished to draw attention. He wished to bring to notice a slight technical modification, in which the central ray was directed laterally through the centre of the plane of the pelvic inlet. This, in the majority of cases, lay along a line joining two external surface points—anteriorly a point about half an inch below the upper margin of the symphysis, and posteriorly that point corresponding to the spinous process of the fourth lumbar vertebra (which, except in obese women, was not difficult to identify). A ray directed laterally through a point corresponding to the junction of the middle and lower thirds of a line joining these anterior and posterior surface markings would bisect the pelvic inlet. From the skiagram obtained with this modified lateral technique the following information might be gathered: (i) the presentation and the position of the fœtal head; (ii) the measurement of the suboccipito-bregmatic or of the biparietal diameter of the fœtal head; (iii) the relationship of the head to the pelvis; (iv) the length and shape of the sacrum; (v) the depth and inclination of the *symphysis pubis*; (vi) the measurement of the antero-posterior dimensions of the pelvis at (*a*) the brim (the *conjugata vera*), (*b*) the mid-pelvis and (*c*) the outlet; (vii) the measurement of the distance between the sacrum and a line joining the ischial spine (the width of the sacro-sciatic notch); (viii) the posterior-sagittal measurement of the outlet.

Dr. Macdonald explained that owing to magnification in the radiographic projection, the diameters as recorded on the film were not the true measurements; he thought it unnecessary to explain how correction was made; it would suffice to say that such correction did not require a profound mathematical scholarship.

Dr. Macdonald went on to say that because the true conjugate diameter shown in such a lateral film was adequate, it could not be assumed that the transverse diameter would be equally so; in such a view one could exclude the flat type of pelvis, but not that type in which the inlet had a transverse diameter less than the true conjugate, and which was termed by Caldwell and Moloy the "anthropoid" type. The transverse diameter was, however, easily computed; if the patient stood erect with the central ray passing horizontally one inch above the *symphysis pubis* at a tube-film distance of five feet, the distance of the transverse diameter from the film was taken as the distance of the centre of the greater trochanters from the middle of the cassette. But Caldwell and Moloy (as well as Herbert Thoms, professor of obstetrics at Yale University, and also a strong sponsor for X-ray pelvimetry) were interested in the shape of the pelvic brim in addition to the antero-posterior and transverse diameters. They required to know whether the widest transverse diameter was nearer to the promontory or to the *symphysis pubis*, and whether the brim was kidney-shaped with the greatest transverse diameter near to the promontory, as in that type which they described as "android". To obtain the true shape of the pelvis it was necessary to place the patient in such a sitting position that the plane of the inlet was parallel to the plane of the horizontal film; the central ray was directed through the mid-point of the pelvis at right angles to the inlet plane. It had to be mentioned that X-ray pelvimetry became more difficult as pregnancy advanced; in late pregnancy one could not obtain the well contrasted black and white film beloved of the cinema director as well as the radiographer.

Dr. Macdonald also demonstrated the technique for pelvimetry of the pelvic outlet, in which information was available on the subpubic angle, the biischial or transverse diameter of the outlet, the depth of the true pelvis, and the inclination, if any, of the inner pelvic walls; such data were of value in diagnosing the male or "funnel" type of pelvis.

Dr. Macdonald concluded by observing that detailed and complete X-ray pelvimetry had in Australia a much more limited application than in Britain or the United States of America; fortunately the pelvic contractions and deformities which were relatively common overseas were seen only infrequently in Australia. On this account Dr. Macdonald believed that, in most cases, the one special lateral film which he had described was sufficient to show whether ample room existed in the pelvis for easy passage of the fœtal head; provided that digital examination raised no doubt concerning the adequacy of the transverse diameter (for example, projecting ischial spines or narrow pubic arch), further radiological pelvimetry was unnecessary.

Hermaphroditism.

DR. R. G. WORCESTER described a case of so-called male pseudohermaphroditism, and DR. H. F. BETTINGER discussed in detail the general biological aspects of hermaphroditism. This

case and comment will be published in full in a later issue of the journal.

Hogben's Pregnancy Test.

DR. H. F. BETTINGER and DR. V. I. KRIEGER described their experiences with Hogben's pregnancy test, which was performed on the South African toad *Xenopus lævis*, and which allowed results to be read within twenty-four hours.

Sarcoma of the Uterus.

Dr. Bettinger also described five cases of sarcoma of the uterus which had occurred at the hospital. The first case was that of a woman, aged forty-one years. Her abdomen had become increasingly swollen during the preceding two years. The menstrual periods were irregular, and flooding alternated with amenorrhœa. On examination a large mass was found in the abdomen; it reached above the umbilicus. At operation several pints of clear brown fluid were present in the peritoneum. Hysterectomy was performed. The uterus contained a number of large intramural fibromyomata and several soft outgrowths were seen at the surface. The histological examination revealed that the tumours partly consisted of the typical whorled arrangement of connective tissue and muscle bundles, but that frequently quite irregular, definitely sarcomatous structures were present. The patient received post-operative deep X-ray treatment. When last she was seen, nine months after operation, she was well and there was no evidence of recurrence.

The second case was that of a woman, aged forty-five years. She had been operated on at another hospital by Dr. W. G Cuscaden. Total hysterectomy was performed, and the specimen was handed over to Dr. Bettinger for examination. The uterus contained a single large tumour. It presented the whorled appearance of a fibromyoma only in a few places; it was mainly soft and often hæmorrhagic. Histological examination revealed a rather mature type of myosarcomatous growth, and invasion of the uterine wall could be clearly demonstrated in the sections. The patient recovered from the operation, but died eight months later with the symptoms and signs of pelvic and abdominal recurrence. A post-mortem examination could not be performed.

The third case was that of a woman, aged fifty-five years, who had been admitted to hospital complaining of a mass in her abdomen and of dribbling incontinence of urine for the last three days. On examination a large tumour with smooth regular surface was felt in the abdomen; it arose about five centimetres out of the pelvis. This tumour was found to be the bladder, containing about 1,000 cubic centimetres of urine. The uterus itself was impacted in the pelvis and removed by operation. It measured 15 by 10 by 10 centimetres, and on the cut surface four nodules of soft friable whitish material in its wall were seen. Dr. Bettinger drew attention to the peculiar histological structure of the tumour. Although it was myosarcomatous in type, the cells showed the "rhythmic" arrangement usually seen in neurinomata (Schwannomata). The patient did not make a complete recovery. She developed signs of a pathological condition in the pelvis, which on account of persistent rises in temperature was taken for an inflammatory process, and deep X-ray therapy was thought to be inadvisable. Death occurred four months after operation. At the post-mortem examination an extensive recurrence in the pelvis was found. Tumour masses had broken through into the rectum. Numerous small nodular metastases were present in the lungs. Their microscopic structure was quite remarkable, in so far as the arrangement of the tumour cells closely imitated the whorled arrangement of a fibromyoma.

The fourth case was of considerable interest. A woman, aged fifty years, was first examined in July, 1939. She complained of irregular bleeding *per vaginam* for two or three months. On examination a uterine fibroid was found protruding through the cervix. This was removed. Histological examination revealed a fibromyoma with œdema, inflammation and hyalinization. Two months later she was readmitted to hospital compiaining of recurrence of the irregular bleeding. A large cervical tumour was found at operation, with a small uterine body on top of it. The histological examination revealed a rather peculiar structure. The main type of the tumour was that of a cellular myoma; but scattered through the tissue numerous large cells with giant nuclei were found. These cells had very little relationship to the surrounding muscle cells, the axis of their nuclei often being at right angles to that of the muscle cells. Apart from these peculiar cells the tumour had such a regular structure that a diagnosis of sarcoma was not warranted. The patient was again seen five months later, complaining of blood-stained discharge *per vaginam* present for one month. On examination the stump of the cervix was found grossly inflamed, the cervix was mobile, the fornices were clear. At operation the cervical stump was removed and it was seen that a tumour of walnut size had developed in it. The histological sections revealed partly cellular myomatous structures without giant cells, but often quite irregular definitely sarcomatous growth. A reexamination of the tumour removed at the second operation disclosed that even in the light of the later developments a diagnosis of sarcoma could not have been made from these sections. The absence of giant cells in the tissue removed at the third operation confirmed the opinion that they were degenerative and not neoplastic in nature. The patient received radon treatment and was again examined seven months after the last operation. The vagina had completely healed; but it was thought probable that abdominal metastases were developing.

Dr. Bettinger went on to say that while these four cases belonged to the group of myosarcomata, the fifth case was of a different nature. A woman, aged fifty years, was admitted to hospital complaining of irregular menstruation for the last two years. Her history was difficult to obtain on account of an emotional complex due to thyreotoxicosis. On examination a large semi-necrotic fibroid tumour was found protruding from the cervix. The tumour was removed at operation. Histological examination revealed that the tumour was composed of closely packed round or short spindle cells. They resembled the cells of the endometrial stroma to such an extent that it was extremely difficult to distinguish a border between the tumour and the covering endometrium. The diagnosis of endometrial sarcoma had therefore to be made. Information was then obtained to the effect that a subtotal hysterectomy had been performed in 1926 and that the part of the uterus then removed had contained a tumour. A comparison of sections from the tumours removed in 1926 and 1940 revealed that their structure was completely identical. Dr. Bettinger pointed out that it was certainly strange that a sarcoma, after a rather incomplete operation, should not recur until fourteen years afterwards. It might be significant from that point of view that Goodall had recently published a paper in which he suggested that these tumours, which had hitherto been designated as sarcomata, were not sarcomata at all, but actually examples of "interstitial endometriosis"; that was, according to him, a non-malignant condition, in which only the endometrial stroma but not the complete endometrium invaded the underlying tissues. Confirmation of his ideas was still lacking, and that single observation did not lend itself well to a critical analysis of his suggestions.

In conclusion, Dr. Bettinger drew attention to the bad prognosis of sarcoma of the uterus. Of the four patients with the myosarcomatous type, two were already dead, one showed evidence of recurrence, and only one had not had a recurrence within a year after operation. The fifth patient also was well one year after operation, but she probably belonged to a different group.

Correspondence.

EXTIRPATION OF THE SYMPATHETIC CERVICAL CHAINS FOR ANGINA PECTORIS.

SIR: Some six years ago you published a letter from me on the immediate results of extirpation of the left and right sympathetic cervical chains, under local anæsthesia, for the relief of pain due to *angina pectoris* decubitus. In this I followed the work of Jonnescu.

Permit me to report the present state of this patient and compare the method and results with that of paravertebral alcoholic block of the first four dorsal sympathetic nerves after the method and technique of White (*Surgery, Gynecology and Obstetrics*, September, 1940).

This patient has for five years back reached an apparently maximum amount of efficiency amounting to being able to drive her car, perform light housework, do light gardening. She suffers from mild discomfort, which rises not infrequently to considerable pain, diffuse and rather vague to locate, being in the chest, neck and over the right eye. Unlike the pain prior to operation, which was relentlessly agonizing and piercing, little relieved by any medical means, the present angina is controlled by morphine, of which drug there are no objective signs. There have been no bizarre signs or symptoms from removal of the three cervical ganglia. Compared with her former state, the operation is partly successful; compared with the more accurate knowledge of the anatomy and physiology of the heart's nerve supply since determined, and newer methods of interrupting the heart's afferent impulses, the results are quite poor.

White's method, which he has so clearly described, I find more hazardous than an open operation. On the cadaver, at

my hands, the lack of resilience of the dead tissues prevents the finer movements of the needle desirable for its correct position. In the patient the more plastic yielding of the tissues, following the different movements of the needle, gives a freer range of movement. At operations, in placing the needle for the first heart nerve (followed by injections of "Novocain", alcohol and finally lipiodol), the subsequent X-ray examination showed the injection deposited below the second, instead of the first, rib on two occasions. The pleura was entered twice, as shown by a coughing reflex. One injection under the first rib resulted in the free flow of bright blood, which, fortunately, stopped on the slight withdrawal of the needle. These mishaps caused no after-effects. I have not observed neuritis in any marked degree from alcohol spread to the intercostal nerves. It is most interesting, and rather dramatic, to see the prompt occurrence of Horner's syndrome, the dryness and redness of the hand on successfully injecting the "Novocain", followed by relief of intractable pain.

Yours, etc.,

E. A. JOSKE.

Balaklava,
South Australia,
December 17, 1941.

THE ASSOCIATION AND THE PUBLIC.

SIR: In your leading article of November 22 the writer states that the general public should be informed of the objects for which the Association stands, and he (or she) is quite correct; but to affirm, as he does, that it is the province of publications such as THE MEDICAL JOURNAL OF AUSTRALIA to counteract this impression and allay public mistrust of this "strongest of trade unions" suggests that he is out of touch with the ways of the public. I am afraid it does not read our journals.

If we want to inform the public of anything, the best way to do it is through the public Press, or one of the other public means of imparting information.

I must admit that before I read the leading article in question I had read a very short paragraph summarizing it in the daily paper; but it did not drive home its message, and the casual reader might easily have been left with the impression that the allegations in Parliament about the power of the Association to withhold registration and to ostracize non-members was the truth.

As a profession we should realize that to work in mystery and secrecy, either in our innermost councils, convenient as this may be at times, or as the whole Association, is to earn the mistrust of the very people we profess to serve.

Yours, etc.,

A. McQUEEN THOMSON.

Barrow Creek,
Northern Territory,
December 17, 1941.

PROTECTION OF PRACTICES SCHEMES AND TAXATION.

SIR: When the war started, the doctors in Tamworth decided to adopt the British Medical Association scheme for the protection of practices of the Tamworth men called up for full-time military duty. There are nine general practitioners in the town, of whom three are on military duty, leaving six of us to do the work normally carried out by nine.

Naturally we are working hard and have not too much leisure. Incidentally the strain is lessened by the abolition of the evening surgery, which is no more necessary than shops opening at night. However, we do not mind the extra work when we feel we are helping to maintain the incomes of the doctors engaged in war work.

Most doctors know the details of the British Medical Association scheme. We work it this way. All incomes for each month, including the military pay of the absent men, is paid into a pool managed by a committee consisting of the two senior doctors and an accountant. The money received is divided up each month. The absent men receive the same percentage of the total income as they enjoyed before being called up, less deductions for expenses which they do not now have to meet. If one of the absent men was getting 20% of the total income he would still receive 20% less the deductions for expenses. If getting 10%, he would still get his 10%. After the absent men were provided for, the balance is divided among the home doctors according to the work done. If A paid in 30% of the total pool money, his share would be 30% of the remainder. If B paid in only 10%, his share would be 10%. But of course A would contribute more than B to the income of the absent men.

Although we had to forgo a large part of the income we earned, it was quite satisfactory because we realized this extra income and work only came to us because the three men were away from their practices. In fact we are only giving to these men what is rightly theirs.

So that though we gave up £304 in October and £495 in November, we considered it only fair and just. Everyone was satisfied.

But recently we received a surprise. The Taxation Commissioner decreed that, irrespective of any pool arrangements, all money coming into the hands of the practising doctors was earned income and the money paid to the absent doctors was declared a voluntary gift. Therefore we would be taxed on the whole money earned by us, even though it is paid into the pool and we receive only a portion.

The position is that we are taxed on income which we do not get. It may pass through our hands, but it goes into the pool and we do not receive it.

It would not matter so much if taxation was on a flat rate, but it increases as the income rises, and as we are doing the work of the absent men our nominal incomes are increased, so the incidence of taxation is higher.

Also all doctors should make provision for the future, so most of us are receiving money from investments. Therefore, with income from investments *plus* the income passing through our hands but paid to the absent men, we are getting up high, with the consequent high taxation.

I should make it clear at this point that we do not for one moment hold that this extra income should not be subject to taxation, but the recipients of the money should pay it. When the scheme was inaugurated they expected to have to pay tax on this money, which is quite separate from their military pay. In fact, they included it when making up their taxation papers.

For the home men to be taxed on income which is paid to the absent men from the pool seems to us absolutely unfair and unreasonable, and we know many Federal parliamentarians agree with us.

But it appears that some medical men in other districts are willing to pay the tax themselves. All I can say is that these men must be only giving up a negligible amount. I question if they are carrying out the British Medical Association scheme as it should be done.

Personally I expect to pay extra tax on £600 or £700 which I will not receive.

I would like it explained by these men why this is fair and equitable. Their attitude has encouraged the taxation authorities to maintain their illogical position, which will cause hardship to those of us doing our duty to our absent colleagues. In fact it will react against the men on active service, because the home men will have to make other arrangements to meet this unexpected increased taxation.

If we deduct the extra tax from the pool it will mean that these men who enlisted believing their families would be provided for as when they were in active practice, will receive very little; probably not enough to meet certain commitments, insurance *et cetera*. Yet the home men must protect themselves.

However, I still have hopes that better counsel will prevail and that the Federal authorities will follow the lead of the State and have this tax paid by the actual recipient, who is willing and expects to pay it.

Yours, etc.,

E. B. FITZPATRICK.

Tamworth,
New South Wales,
December 18, 1941.

A NATIONAL MEDICAL SERVICE.

SIR: Events of the past critical days will probably rescue the above discussion from a purely political controversy, into which I rather feared we were drifting by the trend of published letters.

I hasten to dissociate myself from being classed as a supporter of the present economic system, as such. My argument suggested a modification of the present system of private medical practice, along the lines of a partial subsidy by the Federal Government, to tide us over the war period.

I might add to this the suggestion that all hospitals in Australia might be made available to patients on the basis of a 50% subsidy to the patients towards their fees for hospital service. This would go half-way towards the free service apparently given by the New Zealand system.

I cannot see how a discussion of the relative merits of capitalism and socialism will advance the position of the

medical profession, which is obliged to carry on under either system.

Straight-out government salaries would dry up a large source of medical income, namely private practice, correspondingly restricting the profession to working under governmental supervision—a doubtful advance.

Partial government control by part-time work and part-time salaries has much to commend it as an experimental measure, which could be modified after experience of its practical working.

If the hospitals were opened to all practitioners in active practice, and a better system of post-graduate work instituted, allowing all those who had had some years in practice to obtain practical experience of the later developments whilst their practices were carried on free for them, a big advance would be made.

The honorary system seems overdue for a complete review, and the system of buying and selling practices will no doubt undergo some remarkable modifications in the near future.

In conclusion, I feel that a deeper study of the principles underlying our present method of conducting medical practice will reveal much that will not be corrected by temporary expedients.

Selfishness will be found at the root of many of our modern evils and inequalities, and in so far as we are prepared to substitute service for profit and cooperation for cut-throat competition, in so far will we be able to advance.

We need not fear the rapidly advancing sociological tide, provided the profession is prepared to give adequate service.

Whilst appreciating the general medical service set out by the Federal Council, no one will consider the last word has yet been said on the subject, and some compromise on the whole question may yet be our lot.

Yours, etc.,

H. T. ILLINGWORTH, M.B., Ch.M.

Bruce Rock,
Western Australia,
December 20, 1941.

A SALARIED MEDICAL SERVICE.

SIR: May I congratulate you on the publication in today's journal of the National Health and Medical Research Council's outline of a salaried medical service. Since the appearance of the Federal Council's proposals for a capitation service there has seemed to me to be a grave danger that the profession's representatives, if they felt obliged to accept some change in the basis of medical practice, might take a fateful plunge in the direction of a capitation service without first giving sufficient consideration to the alternatives.

We are all indebted to the Federal Council and its subcommittee for the arduous and disinterested work which they put into the preparation of their scheme. But this feeling of obligation to eminent men and our own laziness in making time to read and think about these matters easily lead us to become engrossed in the details of the capitation scheme to the exclusion of consideration either of the improvement of private practice along cooperative group lines, or of a salaried service. This applies particularly to the district meetings at which the Federal Council's proposals have been considered by the rank and file of the profession. At these meetings a member of Federal Council has usually been present to expound the scheme, and the meetings have felt, probably rightly, that they were called to consider the capitation scheme and could not waste time on alternatives.

I believe that the Federal and Branch Councils would be wise to invite members of the National Health and Medical Research Council to expound their scheme similarly to district British Medical Association meetings. Supporters of a salaried service will have a grievance if their views do not receive equal prominence to those of the believers in a capitation service. In such a grievance would lie seeds of disunity. Whereas, if the alternatives are thoroughly aired and the profession then decides for a capitation service, the minority could be expected to abide by the majority decision, at least for the purpose of presenting the profession's views to the Government and the public.

In any case, this "outline" has apparently been requested by and submitted to the Parliamentary Select Committee on Social Services. In our own interests, therefore, we must consider and criticize it, or we may again be caught napping with no considered reply to a government proposal.

I should like to offer one criticism myself. In the sixth paragraph of Part IV we are told that a recent graduate should "soon qualify for promotion to the grade of Senior General Practitioner". Yet, on counting up, I find that of the 4,203 practitioners in the scheme, 2,335 are junior general practitioners and 417 senior general practitioners. So that a general practitioner could expect, on the average, to spend all except the last seven or so of his years of service in the status of a junior. Alternatively, those destined to become seniors could advance to that grade "soon" after leaving hospitals and the rest (the great majority) remain juniors for life. In that event tact would be the prime qualification for the "senior" in his thirties who found himself directing three "juniors" of fifty or sixty! These calculations are little affected either by a correction for deaths before the retiring age or by the desirable expedient of compelling intending specialists and administrators to spend a few years as junior general practitioners.

I suggest that a longer scale of general practitioner promotions (say three or four grades) would help to solve this difficulty. Without some such change and a modification of salaries to accompany it, there is hardly a single individual who would choose general practice before the more highly salaried practice of a specialty, yet few would deny that we should seek to encourage some of the more able men into general practice. At present many who would otherwise specialize are led into general practice by the economic necessity of an immediate income. The altered grading of general practitioners I am suggesting would also allow of rather more experienced and highly paid men being sent to the "A" and "B" centres, where the isolation imposes heavier responsibilities than many a "junior" fresh from the moral support of a hospital and its consultants would be capable of bearing successfully.

Yours, etc.,

T. DUDLEY HAGGER.

Bingara,
New South Wales,
December 20, 1941.

SIR: In your issue of December 20, 1941, you publish the report of the National Health and Medical Research Council on a scheme for a salaried medical service. I wish to point out that at least one branch of medicine has been entirely neglected in the published scheme, and that branch is anæsthesia.

In the preliminary note of the report on the scheme it is stated that "a great many medical men are overseas with the Australian Imperial Force. It is essential that their interests be fully considered and conserved." Each general hospital serving overseas has on its staff an anæsthetist, and many of these men, when at home, specialize in anæsthesia to the exclusion of all other branches of medicine. Indeed, in the same issue of the journal appears an article from an anæsthetist serving with the overseas forces. Is it intended that these men, as well as those still serving the civil population at home, should be forced to abandon their chosen careers and become physicians with the right to administer anæsthetics?

I am sure that the surgeons who have adopted such specialized branches as, for example, cranial or thoracic surgery will be opposed to the loss of the services of skilled anæsthetists who have devoted their time and energy to acquiring facility in the special anæsthetic techniques called for by these and other branches of surgery.

It is not suggested that all centres would require the services of a full-time anæsthetist or that all anæsthetics should be administered by him; but it is felt that, particularly in teaching hospitals and in hospitals where specialized surgery is to be performed, the employment of the specialist anæsthetist is essential for the advancement of anæsthesia, and that the loss of the full-time specialist anæsthetist would be a retrograde step.

Yours, etc.,

ROBERT H. ORTON,
Acting Secretary, Australian Society of Anæsthetists.

12, Collins Street,
Melbourne,
December 21, 1941.

HERPES RECURRENS.

SIR: I had an opportunity a couple of weeks ago of treating a herpes. I injected the whole vesicular area subcutaneously with "Proctocaine", using about 12 cubic centimetres. Relief was immediate, dramatic and continuous. This information may be of value to Dr. Guy Griffiths.

Yours, etc.,

R. PALMERSTON RUNDLE.

Rockhampton,
Queensland,
December 29, 1941.

Naval, Military and Air Force.

NEWS.

THE following is the copy of a letter written by Dr. Philip D. Wilson, Medical Director of American Hospital in Britain, Limited, to Dr. A. R. Hamilton, Honorary Secretary of the Australian Orthopædic Association. The letter was read at the annual meeting of the Australian Orthopædic Association in Melbourne in October, 1941. We are grateful to the Australian Orthopædic Association for permission to publish it.

I returned from England on January 14th and found your note of the 15th of October awaiting me, asking me to forward in the form of a paper for the 1941 annual meeting of the Association a review of my experiences and the development of orthopædic surgery in the present war. I have been so busy with preparation of other papers that I have not had the opportunity to write, as I should desire, a paper that would comply with your request. On the other hand this invitation is evidently inspired by a desire to obtain the opinions of an American orthopædic surgeon who has recently worked in England and any conclusions he may have been able to draw from his experience that may be of help to the members of your Association in the work that they will undoubtedly have to do in caring for casualties among the Australian Expeditionary Force. I appreciate the honor as well as the opportunity offered by this invitation and I am only too glad to cooperate. I hope that a communication in the form of a letter, although of more informal style than a paper, will serve this purpose equally well.

My opinions are based upon clinical experience in England lasting from September 1st, 1940, to January 2nd, 1941, during which time I was Medical Director in charge of an organization known as the American Hospital in Britain, Ltd. This organization was formed in June, 1940, at the time of the collapse of France, when it was thought that there would be real need for medical assistance for American doctors in England. My contacts were made with the Ministry of Health through the intermediary of Professor Harry Platt of Manchester. As a result of correspondence and cabled exchanges, I set forth on August 22nd with a group of twelve, including five orthopædic surgeons, one general surgeon, one plastic surgeon, three operating theater nurses, one secretary and a technician. We arrived in London on September 7th and were received with every courtesy by the governmental authorities. We were lodged at the Dorchester Hotel as guests of the government and remained there until the 1st of October. Similar courtesies were extended to us by the enemy forces, as the date of our arrival in London corresponded to the opening of the air attack on London and this was continued intensively throughout our stay. Professor Fraser, Director General of the Emergency Medical Service under the Ministry of Health, and his associates cooperated in every way and offered us several sites where we might go to work. We finally elected to install ourselves in the Park Prewett Hospital, outside of Basingstoke in Hampshire. This is a large hospital converted from a former mental hospital by the Emergency Medical Service and now devoted to general medical and surgical purposes. There was a large British staff, including general surgeons, internists, house surgeons and physicians. We were given a block of wards with three hundred beds for our own service. We had also our own separate theater and physical therapy arrangements. We secured quarters by leasing a house outside of the hospital grounds. Park Prewett had the added advantage of a special facial maxillary center directed by Sir Harold Gilies where our plastic surgeon could go to work. Additional personnel was brought over from the United States and our present personnel includes a total of thirty-two persons, comprising twelve doctors, thirteen nurses, four technicians, two secretaries and one orderly. The patients on our orthopædic service were about two-thirds military and one-third civilian. The military cases were chiefly from the army, although there were some from the navy and Royal Air Force as well. Many of these cases were of the type of civil sick, including a few cases of Pott's disease, scoliosis and back pain, a great many injuries and many fractures, the latter chiefly arising from accidents sustained while driving during the blackout. We also had number of the old Dunkirk and Norwegian casualties with compound fractures resulting from projectiles. Some of these cases were very interesting and told incredible stories of how their injuries were received. I remember particularly one man who was a gunner on a destroyer which was dive bombed by an enemy plane. This plane dived too low and when straightening out carried away part of the rigging. A cable loop became twisted around this gunner's waist and he was carried up by the plane a distance of twenty-five feet or more before he was finally dropped, and he sustained severe comminuted fractures of the feet and lower legs. Another man was of much interest because of severe burns about the head and shoulders with frozen lower extremities. He apparently had been dropped into the icy waters of a Norwegian fiord which was covered with flaming gasoline. These men were in excellent spirits, and I have no doubt that the explanation was that they realized they were indeed lucky to still be alive.

Our civilian cases were chiefly air raid casualties. They reached us by evacuation from London or from the couth coast towns and arrived anywhere from a few days to several weeks after injury. Most of these wounds were multiple and severe. I noted that the Thomas splint seemed to be used very little for the transportation of these cases and most of them arrived in plaster. These wounds were uniformly infected, many with gas-producing organisms. Our routine treatment was to explore the wound under general anæsthesia, providing such additional drainage as might be indicated. A dose of from 5 to 7 grams of powdered sulfathiazole was blown into the wound and it was then packed with dry gauze. The fractures, which were generally badly comminuted, were reduced by the use of the Roger Anderson skeletal reduction machine. In the case of the femur, for example, two half pin units were inserted in the upper fragment near the great trochanter and another double half pin unit inserted in the lower fragment at the femoral condyles. The limb was then placed in the machine which grasped the pin units and the screw traction was tightened until shortening had been overcome. Then under X-ray control the fragments were further adjusted in the frontal and sagittal planes until proper alignment was obtained. The pins were then locked in place by the application of a metal side bar that bolted solidly to the two double pin units previously placed in the bone. The entire limb was then encased in plaster of Paris. Since the upper fragment was secured by the pins it was unnecessary to extend the plasters about the trunk in the form of a spica and the hip was not immobilized.

We followed the Orr-Trueta method of closed plaster treatment of the wound and I was greatly impressed with its success. After a varying number of days the temperature usually subsided to approximately normal, the patient was free from pain, his appetite improved and the general condition became excellent. We usually continued sulfathiazole by mouth until such time as the course became afebrile. Dressings were only done at intervals of four to five weeks and then only when the plaster became softened or the odor became too great or there was some other positive indication for intervention. I was quite fearful of this treatment in the case of wounds actively infected with gas organisms, but no disasters occurred and we only amputated one leg because of infection during the whole course of my stay. When gas organisms were present the course of the patient conformed pretty well to the average of the other infected cases. We might find gas organisms still present at the time of the second plaster or they might be absent. They always disappeared in the course of a little time. Of course, these cases will continue to be a problem for many months and my experience was too short to allow me to predict the final outcome. Nevertheless, I am convinced that the closed plaster treatment is ideal for these infected cases. I cannot help but contrast the experience of this war with that of the previous war, where all such cases would have been treated by traction in a Thomas splint and the wounds by the Carrel-Dakin method. All such dressings had to be changed daily and involved a tremendous amount of labor on the part of both doctors and nurses as well as the use of a great deal of dressing material. In addition, these dressings were always painful no matter how gentle one tried to be. In our hospital in England we had one ward of sixty beds which were chiefly compound fractures, and the number of daily dressings would not exceed two or three. The comfort of the patients was the outstanding feature, and while it is true the odor of the plastered limbs was not always comfortable to doctors and nurses, still it could be endured, and the main point is that the patients did well. I believe that the success of this method depends upon several features: first, complete rest and fixation of the wound and of the fracture, second, upon the firm compression of the entire limb with the support that this gives to the circulation, and third, the absence of fresh bacterial contamination of the wound. Recent experiments have shown that pathogenic organisms are showered from a bed when the clothes are turned back and that it is impossible even with the most meticulous technique, to avoid cross contamination of wounds in adjoining beds.

Our plastic surgeon had a most interesting experience working with Sir Harold Gillies. One of the principal problems at the plastic center was that of treating burned

aviators. In the early days of the war these men frequently sustained severe and disabling burns of the face and hands when their plane was set afire in combat and they had to bail out by parachute. Their bodies were protected by their clothing and the face and hands received the chief effect of the flames. In the early days these cases were all treated by the application of tanning solutions, either tannic acid or triple dye. The observations of the plastic surgeons were that this method was harmful and frequently caused the loss of tissue that might have been saved. In the case of the hands the leather-like gauntlets produced by the tanning interfered with the circulation to the distal parts of the fingers and effectively immobilized the fingers in such a way that they became stiff and could not longer be mobilized by any means. As a result of this experience, tanning in the case of face and hands was condemned and forbidden, although in the case of burns of the trunk it was still permitted. Sir Harold and his associates are doing brilliant reconstruction surgery in restoring these men's faces to such condition that they can again appear in human society.

We also had a neurosurgical team working on detached duty in Birmingham. The local neuro-surgeon had been called for army service and no replacement was available until our surgeon arrived. He had a very interesting and profitable experience, but it was chiefly made up of civil conditions and not many military cases.

Our Hospital is going on and I was succeeded by Dr. Wallace Cole, Professor of Orthopædic Surgery at the University of Minnesota, an orthopædic surgeon of wide experience and ability who has gone over to serve six months. We plan to continue the effort and hope when the time of peace finally arrives that something may still remain in permanent form of the American Hospital in Britain.

I cannot close without saying a word or two of the splendid spirit encountered among all the British people, both military and civilian. Many of them were doing not only their work in the day time, but extra volunteer work in connection with air raid protection at night. Women were sharing with the men in this effort and showed no more fear than the men. Everyone put up with the greatest inconvenience, delay and hardship with a smile. I talked with representatives of every class and everywhere the reaction was the same. As for the fear of German invasion, the British to a man hope that it will be tried, and soon. They have absolute confidence in their ability to defend their island home, and this confidence imparts itself to any foreigner who has had the opportunity and privilege of seeing it. These are anxious days for the British and Americans alike.

We have watched the gallant advance of your brave Australians in northern Africa and are hoping that they will be able to stem the tide now in Greece and Yugoslavia. I assure you that your American colleagues in the American Orthopædic Association are with you to a man, and if hopes and good wishes can win the war then it is already done.

National Emergency Measures.

MEDICAL EQUIPMENT.

THE Medical Equipment Control Committee desires to direct the attention of all members of the medical profession to the following information.

Surgical Dressing Gauze and Bandage Gauze.

Surgical dressing gauze and bandage gauze are in short supply throughout the world, and it is therefore imperative that the utmost economy should be exercised in their use. All wounds other than those in the treatment of which gauze packing is essential must be dressed with what is known as a combined dressing. This consists of a pad of wool of appropriate size enclosed in one layer of surgical dressing gauze. This combined dressing may be purchased in the market or made up by hand. Additional pieces of gauze in such a dressing, in the absence of special indications, should not be used.

The use of bandages must be replaced wherever possible by binders, finger-stalls or other methods of retaining dressings in place. All bandages should be cleaned and kept for further use. It is most improper, under present conditions, to cut a bandage unless it is essential in the interest of the patient to do so.

Further information upon the position will be supplied at a later date. In the meantime any purchases of surgical dressings for hoarding purposes, which may be made as a result of the information given above, must be regarded as a most unpatriotic action. If such purchases are observed, the committee will recommend the gazettal of regulations rationing supplies.

Improper Use of Sulphonamides.

The committee has been informed that certain practitioners are using members of the sulphonamide group of drugs, notably sulphapyridine, by inserting the powder at operations into wounds in which there is no reason to anticipate infection. The supply of these drugs is adequate at present to meet all reasonable needs for the treatment of conditions in which they are especially valuable. If the practice of using them in the unnecessary manner described is continued, supplies will be unnecessarily depleted. Under present conditions there is no justification for the use of sulphonamide powders in this way.

An Appeal.

BURSARY FUND IN MEMORY OF STAFF MASSEUSES AT SAINT THOMAS'S HOSPITAL, LONDON.

A WAR memorial fund is being formed in memory of the four staff masseuses who lost their lives when Saint Thomas's Hospital, London, was bombed in September, 1940. A nucleus of the fund has been formed by a generous donation from a former patient, and a small committee has now been set up to decide the form of the memorial.

It is suggested that funds for four bursaries should be raised to help students in the physiotherapy department, to be awarded as follows:

In memory of Barbara Mortimer Thomas (an Australian), primarily for a student from the Dominions.
In memory of Gwendolen Lockyer, primarily for the daughter of a medical practitioner.
In memory of Marguerite Doucet, primarily for the daughter of a past or present member of the services.
In memory of Stephanie Dunn, primarily for the daughter of a clergyman.

All donations will be welcome and should be sent to Miss M. Randell, Massage Department, Massage School War Memorial Fund, Saint Thomas's Hospital, London, S.E.1.

Post-Graduate Work.

COURSE IN SURGERY AT SYDNEY.

THE New South Wales Post-Graduate Committee in Medicine announces that a course in surgery for the M.S. examination, Part II, will be held from March 2 to May 15, 1942. The course will also be suitable for a general revision in surgery. It will be held subject to a minimum of four applications being received. Candidates must notify of their intention to attend the course not later than February 2, 1942. The fee for the course is ten guineas, and further information may be obtained from the Secretary, New South Wales Post-Graduate Committee in Medicine, the Prince Henry Hospital, Little Bay, to whom applications for registration must be made.

Obituary.

EDWARD ELMSLIE BROWN.

WE regret to announce the death of Dr. Edward Elmslie Brown, which occurred on December 13, 1941, at Ipswich, Queensland.

JOHN BALDWIN MEREDITH.

WE regret to announce the death of Dr. John Baldwin Meredith, which occurred on January 1, 1942, at Raymond Terrace, New South Wales.

ANSTRUTHER JOHN CORFE.

We regret to announce the death of Dr. Anstruther John Corfe, which occurred on January 3, 1942, at Glen Innes, New South Wales.

Australian Medical Board Proceedings.

QUEENSLAND.

The undermentioned have been registered, pursuant to the provisions of *The Medical Act*, 1939, of Queensland, as duly qualified medical practitioners:

Burke, William Francis, M.B., B.S., 1941 (Univ. Melbourne), Mater Misericordiæ Hospital, South Brisbane.

Smith, John Beaumaris Dick, M.B., B.S., 1941 (Univ. Sydney), General Hospital, Brisbane.

Stevens, Frank Richard Tod, M.B., B.S., 1937 (Univ. Melbourne), Commonwealth Health Department, Townsville.

Whitehead, Kevin John, M.B., B.S., 1941 (Univ. Melbourne), General Hospital, Brisbane.

Berry, Annie Hayes, M.B., Ch.B., 1937 (Univ. New Zealand), General Hospital, Innisfail.

Macleod, Sadie Catherine, M.B., B.S., 1941 (Univ. Sydney), General Hospital, Brisbane.

Sowter, Reginald Guy, L.R.C.P. (London), M.R.C.S. (England), 1930, Bundaberg Friendly Societies' Medical Institute, Bundaberg.

Nominations and Elections.

The undermentioned have applied for election as members of the New South Wales Branch of the British Medical Association:

Merchant, Lance Verdun, M.B., B.S., 1941 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.

Williams, Owen Upcott, M.B., B.S., 1941 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.

Brand, Nugent Elliot, M.B., B.S., 1941 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.

Waugh, Peter Cedric Phipps, M.B., B.S., 1941 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.

The undermentioned have been elected members of the New South Wales Branch of the British Medical Association:

Deakin, John Henry, M.B., 1939 (Univ. Sydney), 391, Military Road, Mosman.

Hoy, Ronald James, M.B., B.S., 1939 (Univ. Sydney), General Post Office, Sydney.

Kenny, Patrick John, M.B., B.S., 1936 (Univ. Sydney), NX70399, Captain, A.A.M.C., Australian General Hospital, Australian Imperial Force, Abroad.

Kirton, Patricia Anne, M.B., 1938 (Univ. Sydney), c.o. Bank of New South Wales Trust Building, King Street, Sydney.

Klineberg, David, M.B., 1936 (Univ. Sydney), 56, Old South Head Road, Vaucluse.

McLaughlin, William Peter, M.B., B.S., 1939 (Univ. Sydney), 8, Claude Avenue, Cremorne.

Rutherford, James, M.B., B.S., 1941 (Univ. Sydney), Newcastle Hospital, Newcastle.

Paul, James Adrian, M.B., B.S., 1941 (Univ. Sydney), Royal North Shore Hospital, St. Leonards.

Merchant, Lance Verdun, M.B., B.S., 1941 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.

Medical Appointments.

Professor Henry Priestley has been appointed a Member of the Board of Secondary School Studies, in pursuance of the provisions of section 32A of the *University and University Colleges Act*, 1900-1937, of New South Wales.

Dr. Lewis Wibmer Jeffries has been reappointed Director-General of Medical Services pursuant to the provisions of the *Mental Defectives Act*, 1935-1941, of South Australia.

Dr. Constance Alice Finlayson has been reappointed an Official Visitor to the Mental Hospital, Parkside, South Australia.

Books Received.

"A Pocket Medical Dictionary", compiled by L. Dokes, S.R.N., D.N., assisted by T. B. Davie, B.A., M.D., F.R.C.P.; Fifth Edition; 1941. Edinburgh: E. and S. Livingstone. Crown 16mo, pp. 438, with illustrations. Price: 3s. 6d. net.

"Catechism Series: Surgery", Parts I and II, Fifth Edition; 1941. Edinburgh: E. and S. Livingstone. Crown 8vo, pp. 140, with 16 X-ray plates. Price: 1s. 6d. net each part.

"Aids to Pathology", by K. Campbell, O.B.E., M.B., F.R.C.S.; Eighth Edition; 1941. London: Baillière, Tindall and Cox. Foolscap 8vo, pp. 269, with diagrams. Price: 5s. net.

"The Missing Link in Democracy", by F. Trinca; 1941. Melbourne: J. T. Picken and Sons. Royal 8vo, pp. 104, with diagrams.

"Training for Childbirth, from the Mother's Point of View", by M. Randell, S.R.N., S.C.M., T.M.M.G.; Second Edition; 1941. London: J. and A. Churchill Limited; Sydney: Angus and Robertson Limited. Foolscap 4to, pp. 198, with 140 illustrations. Price (English): 10s. 6d. net.

Diary for the Month.

JAN. 13.—Tasmanian Branch, B.M.A.: Branch.
JAN. 30.—Tasmanian Branch, B.M.A.: Council.
JAN. 23.—Queensland Branch, B.M.A.: Council.
JAN. 28.—Victorian Branch, B.M.A.: Council.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Honorary Secretary, 135, Macquarie Street, Sydney): Australian Natives' Association; Ashfield and District United Friendly Societies' Dispensary; Balmain United Friendly Societies' Dispensary; Leichhardt and Petersham United Friendly Societies' Dispensary; Manchester Unity Medical and Dispensing Institute, Oxford Street Sydney; North Sydney Friendly Societies' Dispensary Limited; People's Prudential Assurance Company Limited; Phœnix Mutual Provident Society.

Victorian Branch (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federated Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

Queensland Branch (Honorary Secretary, B.M.A. House, 225, Wickham Terrace, Brisbane, B.17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 178, North Terrace, Adelaide): All Lodge appointments in South Australia; all Contract Practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205, Saint George's Terrace, Perth): Wiluna Hospital; all Contract Practice appointments in Western Australia.

Editorial Notices.

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